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THE VETERINARY JOURNAL

A Monthly Review of Veterinary Science.

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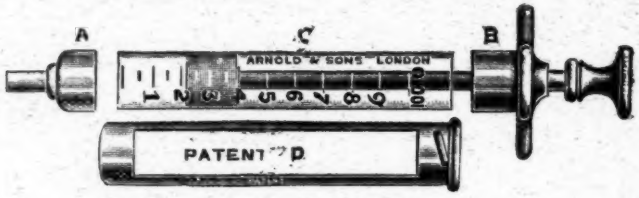

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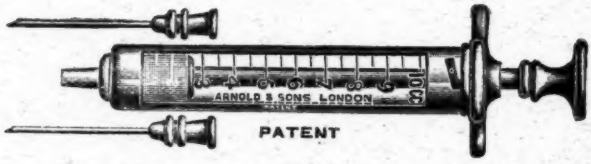

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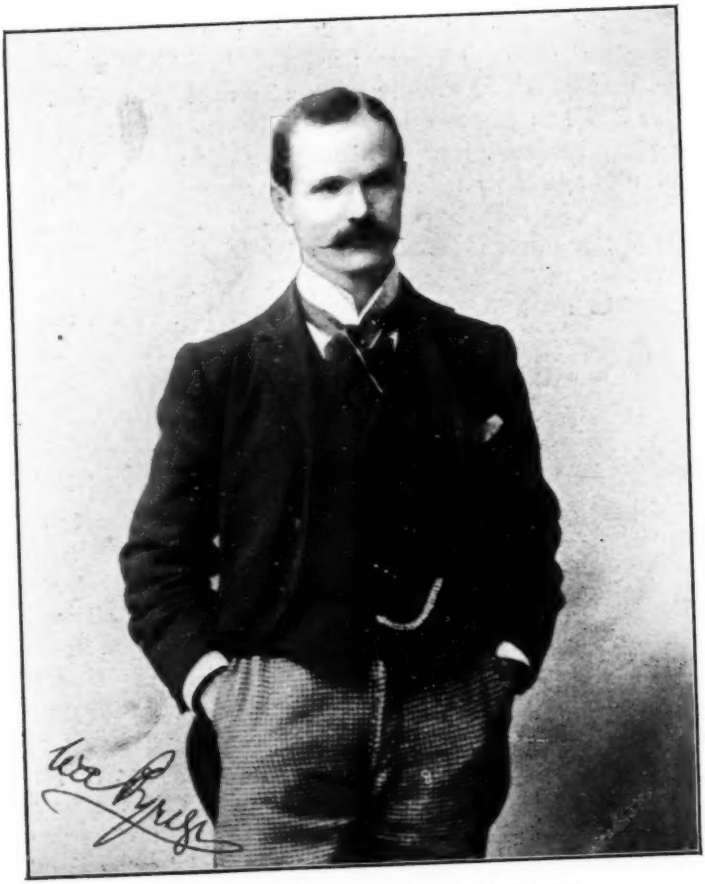

THE LATE MR. W. A. BYRNE, M.R.C.V.S.

THE

VETERINARY JOURNAL

MAY, 1910.

W. A. BYRNE, M.R.C.V.S.

It is with extreme regret that his many friends will have heard of the death of Mr. W. A. Byrne, which occurred at his residence, Castle Strange, Roscommon, on April 17. For some months past Mr. Byrne was in failing health, and had undergone a serious operation, but his death was not so soon expected.

He was yet a young man, only 46 years, and was full of his usual vigour and spirits to the very last. Mr. Byrne was born in co. Roscommon, and received a high classical education. He went through the Agricultural Course at Glasnevin College, and qualified from the Royal Veterinary College, London, in 1889, where he was for some time House Surgeon and Demonstrator in Anatomy, afterwards going to practise in his native county, where he was highly esteemed and respected for his many splendid attributes.

Mr. Byrne took the greatest interest in all matters pertaining to the Veterinary profession, being a member of two Irish associations as well as of the National Veterinary Association. He was for some time a member of the Council of the Royal College, being elected in 1900, and also one of the Governors of the Royal Veterinary College of Ireland. He was one of the regular advisers of the Department of Agriculture in Ireland; one of his very last works was to confer with the Department on the proposed veterinary dispensary scheme for the congested districts. By

Mr. Byrne's death the world of sport has lost an enthusiastic supporter, and the Veterinary profession has lost one of its most able, brilliant, and popular members. His colleagues in Ireland particularly, will miss him, and his place in the ranks will not easily be filled.

Mr. Byrne was an all-round sportsman, a prominent figure in the hunting field, and his colours were often to the front on the Turf. He was a renowed and gifted orator. He took an active part in politics, was an ardent Home Ruler, and had always a deep interest in the welfare of the people. His kindness and generosity to the poor were proverbial, and made him beloved by all who knew him. His local popularity may to some extent be judged from the fact that the funeral cortège was over two miles long. His jovial, amiable manner made him always welcome; he was the best of company, few could equal his telling of a story.

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ndexed B. A. I.

Editorials.

THE STANDARDIZATION OF VETERINARY SERA.

It is really very remarkable what little has been done in the way of attempting to secure uniformity in that increasingly important class of therapeutical agents, such as antitetanic serum, tuberculin, mallein, &c. It is not that uniformity of dose of the finished preparation as sent out by the manufacturers is needed, but some guarantee that the dose shall contain a sufficiency of the essential ingredients required to produce their specific effects. There are many manufacturers of tuberculin and mallein. They can place their products on the market without any guarantee of their accuracy, and that being the case it is little to be wondered at that results of testing with these agents have frequently varied, with a natural loss of confidence in their reliability as testing agents. We fear that those agriculturists who distrust tuberculin have some grounds for their distrust, not of properly prepared tuberculin, but of inferior products. And how can they be distinguished at the present time? It would be a thousand pities that such useful agents should become despised, for if once they become regarded as being unreliable (owing to manufacturers

shortcomings or otherwise), it will be extremely difficult to restore confidence in the minds of the laymen in reference to them.

Then, again, with regard to antitetanic serum. We have frequently heard veterinary surgeons express doubts in no unmeasured terms as to its efficacy. Those doubts have arisen as the result of failure to obtain the specific action attributable to the serum. How can we account for these failures? The answer to this question is furnished in a very excellent paper on pp. 285 *et seq.* by Drs. Moller and Eichhorn. They have tested a number of marketed samples of antitetanic serum, and have found enormous variations in them. Taking the American unit as the standard for comparison, the immunizing dose being 1,500 American units, they found the necessary amount present in some manufacturers' products, but considerably less in others, while in one case there was actually less than one-third the required number of units to produce immunity.

Now these variations can only be remedied by enforcing a definite standard; the products should be guaranteed a definite strength, and any deficiency should be heavily penalized. Such steps are necessary for the welfare of our patients and clients. Moreover, as the investigators referred to say, "It is very essential that the veterinarian should have some assurance of the strength of the antitoxin upon which his standing as a professional man may depend. Under the present conditions the veterinary surgeon has no assurance of the potency of his serum, and is solely dependent on the reliability of the manufacturer."—G.H.W.

COLONIAL VETERINARY SCIENCE.

WE have at last received the somewhat delayed official report of Dr. Theiler's address, delivered at the opening of the session at the Royal Veterinary College of Ireland. The address, however, loses nothing by its delay, and is worthy of careful perusal by all concerned in veterinary education. It has special reference to the conditions under which disease exists and spreads in South Africa, and is certainly most interesting and instructive.

The reference to what is being done by Colonial Governments to assist research is very pleasing. Foreign governments realize

the necessity for substantial grants for this purpose; British Colonial Governments realize it, but England herself lags behind in a woeful fashion. This state of affairs cannot last long. We have often referred to this point, and do not wish to labour it unduly now, especially as we fancy we see a dim light appearing with a promise of something substantial being done in the near future. The Transvaal has built and equipped a laboratory exclusively for the study of animal diseases, at a cost of £80,000, and makes an annual grant of £30,000 towards its maintenance. Is it not time that Britain did a little more out of the National Exchequer instead of crippling research by relying so much on private enterprise?—G.H.W.

"THE BIOCHEMIC HOSPITAL!"

It affords us much satisfaction to give gratuitous and unsolicited publicity to this institution. We have recently received the undermentioned advertisement accompanied by a pamphlet of some twenty pages, setting forth in glowing terms the advantages to be obtained from the "Biochemic" treatment, and a tolerably modest life-history of the enterprising individuals who are responsible for this attractive institution.

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induced to lend any support to such pretensions set forth in such a manner. Certain pages of the pamphlet are amazing, and our readers can, if they desire to take the trouble, obtain them by application, but the final page, which we quote here, shows that those responsible for the "Biochemic Healing System" are fully alive to the importance of adequate financial support. We read as follows :—

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Here is an opportunity for any Member or Fellow of the Royal Veterinary College who feels that the curriculum and experience which have given him professional status and skill are inadequate for his practice, to subscribe his guineas to the society in Streatham and to sit at the feet of the pundits of Eardley Road, S.W., in order to acquire some of the notable lore referred to in their modest booklet. Or perhaps Parliament might be petitioned to accord to the professors of "biochemistry" that recognition which an unfeeling world at present withholds!

General Articles.

THE RELIEF OF ROARING IN HORSES BY EXCISION OF THE LEFT LARYNGEAL VENTRICLE.[1]

By GEO. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.,
Professor in the Royal Veterinary College, London.

At the outset I should like to sound a note of warning concerning the usefulness of this operation. Roaring, in the broad sense, or the production of abnormal inspiratory sounds during forced exercise, may be due to a number of varied causes, and it is not suggested that all and sundry may be relieved by the operation about to be demonstrated. It would be quite useless in cases in which the sound is due to such mechanical obstructions in the air-passages as constrictions of the nostrils or posterior nares, cysts, or polypi, thickening of the pharyngeal and bronchial mucous membranes, and swellings of the guttural pouch. On the other hand, it is likely to be of great service in those cases of what I may term "true" roaring due to paralysis of the laryngeal muscles, which is usually unilateral, affecting the left side.

In order to understand how the desired results are to be brought about, it is first necessary for us to briefly refer to the structure of the larynx and its mechanism. It consists of a cartilaginous framework composed of five pieces, viz., cricoid cartilage, thyroid cartilage, epiglottis, all single and mesially placed, and two laterally placed arytenoid cartilages. They are all movable one upon the other by means of so-called intrinsic muscles, viz., crico-thyroideus, crico-arytenoideus posticus, crico-arytenoideus lateralis, thyro-arytenoideus, and the arytenoideus. The last-named is single and the rest in pairs. They are named according to their attachments, and thus it will be seen that all except the first-named are attached to the arytenoid cartilage and control its movements on its respective side. The vocal cords join the arytenoid cartilages with the thyroid, and they project obliquely into the larynx, with their free edge forwards and downwards, thus forming on each side of the larynx a *cul-de-sac* behind the deep face of the vocal cord and posterior border of the arytenoid cartilage, and which will freely admit one finger in an average horse. The respiratory surface of all the laryngeal structures is lined with mucous membrane.

[1] Read before the Western Counties Veterinary Society prior to demonstrating the operation on several roarers, at Exeter, April 21, 1910.

In normal respiration the arytenoid cartilage moves freely, being drawn outwards by the crico-arytenoideus posticus (the dilator of the larynx) during inspiration, and potentially obliterating the ventricle, while during expiration it dips, as it were, carrying the vocal cord with it, so that the two vocal cords have a tendency towards approximation. This action is due to the constrictors of the larynx. In unilateral paralysis of the opened larynx this movement can be readily seen on the normal side, whereas on the other side the arytenoid cartilage and vocal cord are motionless beyond slight vibration, *i.e.*, the normal rhythm is wanting on that side. The effect of this on the horse is that the larynx is not properly dilated during inspiration, and if exercise is forced some degree of distress occurs, and the flaccidity of the vocal cord allows it and the arytenoid to vibrate and produce the sounds known as roaring or whistling.

I do not intend to discuss the etiology of the condition now, but the fact that the paralysis and atrophy are so commonly limited to the muscles supplied by the left recurrent nerve renders it reasonable to conclude that the peculiarities of the course of the nerve in the thorax are responsible.

Obviously the simplest way of overcoming the distress is to insert a tracheotomy tube, but that is always unsightly, wants constant daily attention, and there is always a considerable amount of noise remaining. To do away with these objections several operations on the larynx have been suggested and attempted. The pioneer appears to have been Professor Günther, of Hanover, who began experimenting in 1834, and practised at various times the following operations, according to Liautard: (1) Resection of both vocal cords; (2) removal of the vocal cord of the paralysed side of the larynx; (3) the partial excision of the arytenoid cartilage; (4) the total extirpation of the arytenoid (arytenoidectomy); (5) the removal of the vocal cord and of the corresponding laryngeal ventricle; and (6) the fixation of the arytenoid cartilage by an ankylosis at its articulation with the thyroid cartilage.

According to Fleming, Professor Günther "finally endeavoured to bring about a firm adhesion between the inner surface of the thyroid and the outer surface of the arytenoid cartilage by excising the laryngeal sac, but leaving the vocal cord intact." This is the operation we propose to perform to-day. It has lately been reintroduced by Professor W. L. Williams, of New York, who worked it out quite independently of Günther's work. He claims very encouraging results, and kindly described it to me and showed me excellent photographs

of larynges which had been operated upon with success, in which the arytenoid cartilage and the vocal cord were adherent to the side of the larynx. He subsequently operated in Hobday's presence, and since then I have also seen Hobday operating. The present popularity of the operation is undoubtedly due to Hobday's enthusiasm.

Still another operation has been attempted, and fair success claimed—viz., arytenoidorraphy, which consists of suturing the vocal process of the arytenoid back to the wing of the thyroid, and so preventing the dipping and vibration.

It seems to me that the operation we are about to perform is likely to be fraught with the fewest complications, and, as many horses are now being operated upon, we shall soon know what results to expect. It must only be performed on cases with undoubted paralysis, and care must be taken that no cartilages are wounded beyond the original incision through the cricoid.

The Operation.—The horse must be cast on his side and chloroformed. While being anæsthetized the laryngeal region may be washed, and then the patient must be fixed on his back with his head resting straight on his poll, and the nose fully extended. I usually inject adrenalin or renastyptin under the skin and into the muscles of the operation area in order to diminish the oozing of blood which otherwise may obscure the deeper operation field. An area is then shaved on the middle line, 2 in. wide, extending from the intermaxillary space to at least 6 in. behind the level of the angles of the jaws. After disinfection the incision is made about 4 or 5 in. long, the anterior limit being level with the angles of the jaw, and cutting through the skin at one stroke. A number of cutaneous vessels must now be picked up with artery forceps. Next cut the paniculus and expose the sterno-thyrohyoideus muscles. The junction of these along the middle line can be seen, and the incision is continued through this septum down, to expose the body of the thyroid cartilage, the cricoid cartilage, and one tracheal ring. The danger from carelessness lies on either side of the seat of operation where the thyro-laryngeal artery comes from the carotid. Another danger zone is the anterior limit of the cutaneous wound where the submaxillary glands are situated, and with the submaxillary artery, and vein and Stenson's duct on either side.

The skin and muscles are slightly retracted, and the larynx palpated to definitely locate the thyroid cartilage, cricoid cartilage, crico-thyroid ligament, and the crico-tracheal ligament. With the head perfectly straight, the crico-thyroid ligament is pierced and the incision

extended forward to, but not including, the body of the thyroid cartilage, and then backwards, cutting outwards through the cricoid cartilage and the crico-laryngeal ligament. I have not found it necessary to cut the first tracheal ring, but that may be done. I have not found any necessity to use a tampon cannula. The larynx is now held open by means of a special spreader and the interior examined. If one or both arytenoid cartilages lies motionless, the operation may be proceeded with, but if both are moving freely during respiration it is useless to proceed, since paralysis is obviously not the cause of the trouble.

Having decided to proceed, the vocal cord of the paralysed side, which is noticed towards the anterior extremity of the wound, must be drawn back by a small repeller, and the entrance to the ventricle revealed. A circular incision is then made through the mucous membrane with a razor knife, and both edges of the portion to be removed are grasped with long forceps. The membrane must then be separated carefully with a blunt spatula or a finger all round and to the bottom of the sac and then lifted out. It is somewhat thin at the depth and easily torn there, but it is rather thick for the rest of its extent. If great care is not taken, a piece of the *processus vocalis* of the arytenoid will be removed also. All that now remains is to cleanse the larynx and trachea of blood and to mop out the ventricle. The horse is rolled on to his side and allowed up as soon as possible. No sutures are required or advisable. Subsequent treatment consists of sponging the lips of the external wound several times daily with damp wool wrung out in carbolic solution.

For between two and three weeks the horse breathes through the operation wound and his nose, and during that time the connective tissue surfaces of the ventricle are becoming united, the arytenoid cartilage and vocal cord being bound down in course of time to the side of the larynx. The external wound should be completely cicatrized in four weeks, but the ventricular cicatrized tissue is not strong enough to warrant galloping the horse as a test for at least eight weeks. In other words, the horse must have absolute rest from exercise for four weeks, and must then undergo a course of conditioning, and not thoughtlessly galloped for another month. Hence the best time is the end of the hunting season for hunters.

Now as to the usefulness of the operation. In the first place, it is an excellent substitute and improvement on tracheotomy. It is not likely to make an unsound horse sound. Unworkable horses will be made workable, and a hunter roarer will not be heard "three fields

away," as the saying goes. It will not affect the heredity of the condition either on the dam's side or on the part of the sire. In fact, I go so far as to say it ought not to be performed on a sire, otherwise it is countenancing a fraud.

In a nutshell, distress should be relieved, and sound either much reduced or completely obliterated. It remains to be seen yet in what percentage of cases these desirable results will be obtained. The operation, however, would appear very promising, and there is still the consolation of knowing that if the required relief is not obtained tracheotomy may still be resorted to.

Possible ill-effects are: Pneumonia, asphyxia, excessive granulation, and ordinary risks of casting and of wounds, as tetanus and malignant œdema, &c.

Indexed By Ala

A DISEASE OF SHEEP[1] IN TASMANIA.[2]

By J. A. GILRUTH, D.V.Sc., M.R.C.V.S., F.R.S.E.
Professor of Veterinary Pathology in the University of Melbourne.

I HAVE the honour to report on the result of my investigations regarding an extremely fatal disease of sheep which appears annually in certain districts of Tasmania, and some cases of which I was able to examine, in company with Dr. Willmot, Government Veterinarian, during my recent visit.

History.—The disease has apparently been known for a number of years, and Dr. Willmot is of opinion that its occurrence is more general than is at present recognized.

It occurs as a rule at a definite period of the year, viz., during the months of August and September. It is associated, not so much, however, with the actual time of the year, as with the condition of the herbage about that season.[3] According to the observations of those who have had considerable experience of its ravages, it prevails just immediately after the early spring rains, when the young grass is

[1] As it is evident this disease requires a special name—one which, while being indicative of its chief characteristics, would at the same time convey a suggestion of its similarity to other diseases of somewhat allied character—I consulted my colleague (Professor Allen), to whom I am indebted for the valuable suggestion of "Malignant Transudation." I therefore propose it should be known by this term, and that the pathogenic bacterium be designated the *Bacillus transudationis maligna*.

[2] A Report to the Minister for Agriculture, Tasmania.

[3] It is interesting to note that sheep display no definite immunity to experimental inoculation with the specific microbe during the summer months as has been demonstrated by a number of experiments I have conducted since writing this report.

beginning to shoot through the ground. Hence, owing to the mild weather and copious rains experienced this year during the month of June, it was fully anticipated by those who have experience of the disease that outbreaks would occur much earlier than usual, and these fears were justified.

Soil.—Beyond the fact that animals do not appear to become affected on the poorer classes of soil, no definite information could be elicited.

Animals Affected.—The disease is confined to sheep, and particularly to young sheep (hoggets) in good condition. It seldom or never attacks adult sheep, and has never been known to affect even hoggets in comparatively poor condition or suffering from diarrhœa, &c.

That the disease is a serious one, both for the flock-owner and for the State, is proved by the fact that on the property which I visited in connection with the investigation the mortality amongst hoggets from it alone is from 10 to 15 per cent. annually. If its occurrence is at all widespread, the necessity for preventive measures being taken is therefore manifest.

Symptoms.—These may be summed up in the one word "None." The owner of the property visited informed us that during his ten years' experience of its ravages he had only observed two live animals affected with the disease, and, curiously enough, one of these cases occurred on the date of our visit. The almost universal experience is for the shepherd to find one or more animals lying dead. There is no evidence of struggling prior to death, the position assumed being generally that of sleep. Usually, however, there is considerable distension of the belly through generation of gases within the stomachs and bowels, and, in addition, the whole of the carcase is in a more or less advanced stage of putrefaction, while there is commonly a blood-stained frothy material oozing from the nostrils. The skin of the thighs and armpits is generally dark and livid in colour and puffy to the touch, while the wool can often be readily removed. On opening the carcase there is generally a decided odour of putrefaction, and more or less blood-tinged fluid around the bowels and in the chest cavity. Such, shortly, are the features of the disease, as they have been noted by careful observers.

As sick animals are so seldom seen, no data can be acquired as to the symptoms ordinarily exhibited. Judging from the living case I observed, however, and the experiments conducted, there would be lassitude, loss of appetite, moping, &c., for some hours before death

actually occurred. Death seems more prone, even in experimental animals, to occur during the night or early morning, which, together with its rapid progress and with the difficulty of carefully examining individual sheep in a flock, explains the absence of any definite information.

Cases Examined.—On the morning of the day previous to our arrival two hoggets were found dead; while on the day of our visit another death had occurred, and one hogget was evidently suffering from some serious disease, and on the point of death.

The three carcases, which had been kept for examination, were extremely distended with gas, showed bloody froth at the nostrils, and were in advanced stages of decomposition; the two, which had been dead for nearly thirty-six hours, too much so for examination to be attended with any degree of satisfaction.

The animal which died during the previous night or early morning was, however, carefully examined *post mortem*, specimens of fluids and tissues being secured for more detailed microscopic and bacteriological examination later on. Following are the notes on the pathological changes:—

Carcase well nourished and in fat condition. Body still retaining some warmth in the armpits, &c. *Rigor mortis* fairly well established. Extreme tympany of the stomachs and intestines, the abdomen being greatly distended. Bloody, frothy material issuing from the nostrils, and evidently arising greatly, if not solely, from the decomposition and the tympanitic condition. No evidence of diarrhœa. No discoloration of the skin. Wool not markedly loose. (In the other carcases discoloration of the skin of thighs, &c., was marked, and probably of *post-mortem* origin.) Underneath the skin of the abdomen and thorax was a quantity of dirty-looking fluid exudate. Abdominal cavity: About a pint of dirty serous effusion, with a distinctly putrid, but peculiar, odour. The stomachs were apparently normal, but for *post-mortem changes* and *post-mortem* digestion of the mucous lining of the abomasum. The intestine, besides some *post-mortem* changes, showed injection of the peritoneal small blood-vessels and patches of hæmorrhage; the rectum contained normal fæces. Liver: Discoloration, due to putrefaction on posterior border; the substance was congested, and a few, small, irregular, greyish necrotic patches were present under the capsule. The kidneys: Pulpy, intensely congested, and broken down with gas. Spleen slightly enlarged, pulpy, and containing gas. Thorax: The cavity contained about 10 oz. of blood-tinged serous fluid, with comparatively little putrefactive odour. The lungs were normal. The pericardium

contained several ounces of effusion similar to that within the pleural cavity. The heart was normal, and the blood feebly coagulated, but did not emit any offensive odour.

The live sheep, which was very weak, dull, disinclined to move even when forced, and barely able to stand, was then killed for examination.

Post-mortem Examination.—Animal fat. Underneath skin of abdomen a small quantity of clear effusion. Abdomen: The cavity contained about a pint of slightly turbid liquid. The intestines from the stomach to the floating colon were practically empty, but normal. The rectum contained normal fæces. Abomasum (fourth stomach) practically empty. At the greater curvature near the cardiac extremity an irregular area of about 2 sq. in. of the wall was thickened and inflamed. On the mucous surface the greater part of this area was in a state of necrosis, the lesion presenting the appearance of a dirty, greyish irregular patch surrounded by a zone of dark-red, swollen mucous membrane.[1] On section the wall was found to be markedly thickened, and the tissues underlying the mucous surface very œdematous. The inflammation extended through to the peritoneal (external) covering of the stomach. The other abdominal organs were normal, the kidneys only being congested. Thorax: The cavity contained about 8 oz. or 10 oz. of clear serous fluid, which rapidly became of a jelly-like consistence after exposure. The pericardium contained a small quantity of similar fluid. The surface (epicardium) of the heart was injected and showed one or two small hæmorrhagic areas, but the organ was otherwise normal. The lungs were normal.

These *post-mortem* examinations, together with the general history of the disease, and its incidence as regards the condition and age of the affected animals, as well as the seasonal occurrence of the outbreak, led me to form the tentative opinion that the malady was similar to, if not identical with, a disease affecting hoggets in New Zealand, which I had already investigated; and also of a similar nature to that disease of sheep known as Braxy, in Scotland. Subsequent examination and experiments have confirmed this opinion.

During the conduct of these *post-mortem* examinations, specimens of blood effusions, &c., were secured in sterilized pipettes for bacteriological examination; smears were made directly for microscopic determination of the bacterial contents at the time of *post-mortem* examination; and portions of tissues were also preserved.

[1] Subsequent microscopic examinaton (*see* addendum) proved this to be the original esion and the location of multitudes of the apparently specific bacterium.

On my return to Melbourne these materials were carefully examined, and attempts made by experiment, &c., to ascertain the exact nature of the disease and isolate its determining factor. Success has attended these efforts. Unfortunately, owing to the lapse of time between death and the *post-mortem* examination of the first case, a large number of bacteria, obviously of foreign or accidental origin, had invaded the fluids and tissues, and multiplied greatly: Even in the animal which was slaughtered these accidental microbes had been able to traverse the intestinal wall during the comparatively prolonged illness, and complicate the position so far as bacteriological examination was concerned. The contents of the intestine, particularly of the large bowel, are in all animals found to contain enormous numbers of bacteria, including those of a putrefactive nature, and immediately after death (sometimes before that occurs), numbers of these organisms penetrate the wall, enter the blood-stream, the abdominal cavity, &c., and multiply with very great rapidity. The consequence in this instance was that an enormous amount of work had to be done before the specific bacillus causing the disease under investigation could be finally isolated and cultivated in a state of purity, as can be seen by reference to the addendum. However, already, although there remains much work of scientific and even practical interest to be accomplished, we have sufficient data to enable a fairly complete account of the nature, cause, and pathology of the disease to be rendered.

Nature of Disease.

The disease is characterized by an intense toxæmia or general poisoning of the animal's system by means of the toxic products elaborated by the specific bacterium during its multiplication within the animal body, accompanied by the formation of serous exudates or effusions (more or less blood-tinged) within the serous cavities and subcutaneous tissues, congestion of the kidneys and liver (probably as a result of the attempt by these organs to neutralize and eliminate the toxic products), often necrosis of small areas of liver substance, and occasionally congestion of the spleen, hæmorrhages about the heart, &c. Either before or soon after death there is hæmolysis or disintegration of the red blood-corpuscles throughout the body, and putrefactive changes may either accompany the terminal phases of the disease, or rapidly follow death, owing to the invasion by putrefactive organisms from the intestine.

The disease is due to a bacillus which, on being introduced into the connective tissues, such as that underneath the skin, and probably that

underneath the mucosa of the alimentary canal, multiplies with great rapidity, and generally under natural conditions produces death so rapidly that premonitory symptoms are rarely observed.

It is possible that the bacillus may be introduced into the tissues under natural conditions in two ways: (1) by pricks, &c., of the skin, with contaminated vegetable spines or prickles; and (2) through abrasions in the wall of the alimentary canal made by parasites, &c.

While it is impossible to deny that natural inoculation could occur by the first means in view of the experiments detailed, it must be admitted that the season of the year, the bareness of the pastures, and the state of the herbage, are against the probabilities of its being a common occurrence. On the other hand, the second means is likely to be common: The micro-organism undoubtedly has its natural habitat in the ground; the young blades of grass shooting through after rain often carry considerable portions of earth; the sheep, being a close-grazing animal, is very liable, therefore, to ingest particles of bacteria-infected soil before natural germicidal agencies, such as sunlight, have had time to operate; the common presence of parasites of various kinds in the stomach and intestines of sheep, especially at the period of the year when the disease prevails, provide a ready means of abrading the mucous membrane, and so facilitating the entrance of the bacillus to the adjacent tissues; the early onset of putrefactive changes following death in natural cases, compared with cases where the bacillus has been experimentally introduced under the skin (*i.e.*, much further away from sources of contamination); and, above all, the definite lesion of the stomach, together with the very small nematode found in the sheep which was killed (*see* results of microscopical examination), all support this view.

Adult sheep appear to be immune, or almost immune, to this disease. This has not yet been demonstrated to be the case by experiments, but it will be observed that the two eighteen-month-old sheep survived much longer than the eight-month-old sheep the introduction subcutaneously of exactly the same-sized dose of the same culture.

Furthermore, the disease does not appear to affect naturally sheep in poor condition, or, indeed, sheep which are not in good fat condition. In these respects this disease, as in many others, simulates blackleg in cattle, which rarely attacks naturally any but young stock in very good condition, and can be even experimentally but rarely conveyed to calves in poor condition. The disease is, however, quite distinct from blackleg, although it may be classed under the same group. The blackleg bacillus is innocuous for rabbits and pigeons, whereas the bacillus of this disease is extremely fatal (*see* experiments).

Infection.—The disease is neither infectious nor contagious in the ordinary sense. It is unlikely that it is ever transmitted from an affected living sheep to a healthy animal.[1] The bacillus, like those of blackleg, anthrax, and malignant œdema, probably has its normal life in the ground, and so to say, only becomes parasitic by accident. While this is true there is no doubt that the carcase of an animal dead of the disease must become a potent source of fresh infection, In our studies we have observed that although at the moment of death the blood of an animal may be apparently free of bacilli so far as microscopical evidence can determine, yet if kept for some hours at blood heat the few which must have been present—perhaps only one bacillus to several drops—have multiplied so rapidly that they may, on again examining the blood microscopically, be found in great numbers. A fat sheep retains after death, even in cold weather, its body warmth a sufficient length of time to enable the bacilli to multiply enormously, and render each drop of blood a possible source of contamination. These bacilli form spores which, like all bacterial spores, are extremely resistant to heat, especially dry heat, and other natural germicidal agencies. So long as a carcase remained unskinned there would be probably little danger of spreading the germs; but I presume the skin is generally removed. Then, if the carcase be not deeply buried (or, better, burned), these myriads of spores are liable to be spread by dogs and other carrion animals over a considerable area and new centres of infection (endemic) established. In connection with this I am pleased to record that the gentleman whose property I visited assured me that he is always careful that all dead animals are buried; but for this precaution his losses might possibly have been heavier.

Characters of the Bacillus.—A short rod often found in pairs, non-motile, sporulating generally at one end but often in the centre, Gram positive, strictly anaerobic, growing readily in blood-serum, and in artificial media, to which serum is added, when distinctly alkaline, and air excluded rigorously, but refusing to grow in neutral or acid artificial media.

In size it is fairly constant, being from 4 to 6 μ in length, and 0·5 μ in breadth.

[1] The absence of contagion in the ordinary sense was shown by the following experiment conducted later: A ewe, with lamb three months old at foot, was placed in a pen. The ewe was inoculated with a culture of the bacillus, and succumbed in sixty hours. The lamb remained in the same pen for a fortnight, without exhibiting any evidence of illness. At the end of that period it was also inoculated with a culture, and succumbed in due course. Thus the infection was not conveyed by contiguity, and immunity was not acquired by the lamb.

Red blood-corpuscles of all animals become rapidly dissolved as a result of the growth of the bacillus. Serum of sheep tinged with blood becomes rapidly turbid, the blood-corpuscles dissolved, and the liquid early acquires the same dirty-looking tint that characterizes generally the peritoneal effusions in sheep.

The bacillus in growing produces a small quantity of gas in serum, and in ordinary alkaline serum-broth, with a slight colour that cannot be termed strictly putrefactive, though it in some measure suggests such a description.

In small doses the bacillus is rapidly fatal to the guinea-pig, the rabbit, the pigeon, the rat, and to the sheep. In young sheep under a year old death occurs in from eighteen hours (with a dose of $\frac{1}{2}$ c.c.) to thirty-six hours (with $\frac{1}{4}$ c.c.) of a culture a few hours old. In older sheep eighteen months old, even with the smaller doses, death occurs within forty-eight to sixty hours.

Post-mortem Appearances.—All animals present practically the same *post-mortem* picture. There is the œdematous subcutaneous swelling of the inoculated region, more or less peritoneal, pleural, and pericardial effusion often somewhat blood-tinged or turbid, injection of the peritoneal and intestinal vessels, congestion of the kidney and liver with often areas of necrosis on the latter, and often small hæmorrhages about the heart, the severity of the lesions depending greatly on the period elapsing between inoculation and death.

When a naturally affected animal is found dead, there is always bloody froth issuing from the nostrils, extreme distension of the belly with gas, and often more or less discoloration of the skin of the inner part of the thighs. On skinning the carcase the tissues underneath the skin of the belly are infiltrated with a blood-tinged watery material; there is more or less gas in the muscles, &c.; and a putrid odour is usually noticeable, sometimes very marked. The cavities of the belly and chest and that enclosing the heart contain a quantity, varying in amount, of turbid, dirty-looking fluid; the spleen, liver, and kidneys are more or less easily broken down, and contain gas bubbles; while the heart shows hæmorrhages, small in extent, either beneath the covering or lining membrane.

As already stated, the putrefactive odour and the gas bubbles are chiefly the result of the entrance of putrefactive germs through the intestinal wall into the cavity of the abdomen and the blood-channels. This was definitely shown to be the case in the *post-mortem* examination of sheep 2 in the experiments.

Prevention.—While it is more than probable further research (which

would require much time and expenditure of money) would discover a system of preventive inoculation similar to that adopted with so much success in blackleg and in anthrax, yet, meanwhile, some practicable measures can be suggested that have the commendation, at all events, of being inexpensive. As a matter of fact, from my experience of a similar, if not identical, disease in New Zealand, I tentatively suggested certain measures to the owner of the property visited.

The first of these obviously consists of the burning, if possible, and, if not, of the burial, of any carcase where it is found, in order to prevent the contamination of fresh centres by natural agencies, such as dogs, running surface-water, &c.

The second is based upon the seasonal nature of the disease and the age incidence, and is as follows:—When late winter or early spring rains occur, especially if the weather is mild and the result is likely to be a flush of young grass, the young sheep should be removed to the rougher pastures (even at the expense of some loss in condition). The older sheep—for example, the ewes—could be placed on the affected or suspected pastures if found necessary in the management of the farm. When the young grass had attained a considerable growth, the younger sheep could be again placed in the paddocks in question, care being exercised to see that at no subsequent time during the danger months was the feed eaten close down. The disease of a similar nature in New Zealand affecting only hoggets in winter fed on turnips does not appear till the roots are well eaten down, or until the sheep are liable to ingest more or less soil along with the food, in which respect it also resembles the disease under review.

Similar Diseases and Comparisons.

Anthrax.—Is similar in the suddenness of death without previously observed symptoms; but the almost constant enlargement of the spleen, its pulpy tarry nature, and the tarry condition of the blood are marked naked-eye differences; while under the microscope the characteristic bacillus could not be mistaken for the bacillus of this disease.

Blackleg.—While blackleg is said to occasionally attack sheep—and experimentally they are very susceptible—in my experience it has never occurred naturally, even on farms where numbers of cattle die annually. The characteristic swelling of an affected group of muscles (those of the hind-quarter, fore-quarter, loin, &c.), the crackling feel of the skin even during life (so marked in blackleg), and the characteristic smell of blackleg-affected muscles, are absent. The blackleg

bacillus is larger, although otherwise very similar; but it is innocuous for rats, rabbits, pigeons, &c.

Malignant Œdema.—This disease in certain countries is common in sheep as a form of "blood-poisoning," through infection of wounds after shearing and "marking."

There is great swelling and blackness of the tissues in the vicinity of the wounds, with much gas-formation even during life, which, along with the time of occurrence, renders a distinction easy. The specific micro-organism is quite different in certain characteristics, and could not be mistaken for that of the present disease.

Braxy.—A disease common in Scotland and North-western Europe, Iceland, &c., closely resembles that under review, in its seasonal and age incidence, the susceptibility of fat animals, the absence of *ante-mortem* symptoms as a rule, the rapid putrefaction following immediately on death, the presence of stomach lesions, peritoneal and other effusions, and, generally speaking, the *post-mortem* appearances. The bacillus isolated by Hamilton in Scotland (see Report of Departmental Committee on Louping Ill, and Braxy, 1906), however, though presenting a number of points of similarity, yet differs in many respects, chiefly in regard to odour of cultures, virulence of cultures for guinea, pigs, and even for sheep.

"Braxy-like" Disease of Sheep in New Zealand.—So provisionally termed by me because of its great similarity in age and condition of affected animals, the seasonal incidence, absense of symptoms, and *post-mortem* appearances, to true braxy, is the disease which most closely resembles, if it is not identical with, the affection in Tasmania. Owing to the success of the preventive measures recommended rendering it impossible to secure material for completing certain details of the investigation, which had been postponed pending the erection of new experimental buildings (a circumstance fortunate for the farmer, but rather disappointing to the investigator), I am unable to compare the two diseases as fully as I should desire.

Summary.

The disease belongs to what has been designated the Braxy group.

It is due to a specific microbe, a bacillus, which forms resistant spores, and hence is able to retain its virulence in the soil for a long period.

The bacillus may gain entrance to the system of the sheep either by means of skin punctures or through abrasions in the lining membrane of the alimentary track.

Rarely are any symptoms of illness observed, affected animals being usually found dead in the morning.

As putrefaction follows rapidly on death, the carcase is almost invariably discoloured, greatly distended with gas, and a greater or less quantity of blood-tinged froth is seen issuing from the nostrils.

On opening the carcase there is nearly always present a quantity of fluid, dirty-looking, or more or less blood-tinged, in the belly and chest cavities, and also in the sac enclosing the heart. The disease affects principally hoggets, and is almost confined to those in good condition.

It is most commonly seen during August and September, but may appear earlier in the year, and seems to be definitely associated with the spring growth of young grass.

The disease is not contagious in the ordinary acceptation of the term, the common source of infection being the ground.

Dead carcases should, however, be buried deep, or preferably burned, as such are otherwise liable to reinforce the infection and establish new areas.

Preventive measures at present available are (1) destruction of carcases, and (2) removal of hoggets from infected pastures during the danger season of the year.

Recommendations.—I suggest that full particulars regarding the disease should be circulated by you amongst the sheep farmers in the State, and that they be requested to co-operate with the Department of Agriculture, both in ascertaining its extent and in diminishing the losses sustained.

All measures of quarantine and restriction of movement should be eschewed, as they are unnecessary, and would in any case prove futile. I feel certain that, as elsewhere in my experience, were the farmer to feel confident that his name would not be divulged, directly or indirectly, and so the reputation of his property be unaffected, he would welcome any assistance by the State, and furthermore would freely co-operate in the rendering of all information regarding this and similar diseases that may from time to time afflict his stock.

To enable the farmer to afford the necessary assistance in a concise form it would be well to attach to the leaflet a perforated page containing the following queries, leaving spaces for answers:—

(1) How long have you been in possession of your property?
(2) When did this disease first make its appearance?
(3) What is your annual percentage of mortality?
(4) What breed and age of sheep do you find most affected?

(5) Is any special class of soil affected?

(6) At what period of the year do the losses usually occur?

(7) Has your annual loss increased or diminished since the disease first appeared?

(8) Kindly state any other information which you think would be of value.

(The addendum, which contains the details of the Bacteriological Investigations, is unavoidably held over until next month.—EDS., *V. J.*).

THE INFLUENCE OF OÖPHORECTOMY UPON THE GROWTH OF THE PELVIS.[1]

BY S. G. SHATTOCK AND C. G SELIGMANN.[2]

WHEN we recall the widespread and diverse effects induced by early castration upon the growth of the accessory organs of generation—the prostate, the vesiculæ seminales, the Cowper's glands, not to mention the arrest or modification resulting in the secondary or external sexual characters—we are naturally led to ask how far removal of the ovaries in the young is followed by a corresponding hypoplasia or undergrowth in the accessory parts of the generative apparatus in the female. These parts comprise not only the uterus, vagina, and mammary glands, with the glands of Bartholin, but also the pelvis, and it is with the last-named that the present communication deals.

THE INFLUENCE OF DOUBLE OÖPHORECTOMY UPON THE GROWTH OF THE PELVIS.[3]

That the growth of bone may be modified by the absence of certain internal secretions appears from some of the results of castration and of disease. After castration, carried out in the young of the Herdwick sheep, a variety in which the male alone is furnished with horns, not only is the growth of the horns (as we have found) completely prevented, but, in addition to this, the normal change of form which the male skull undergoes no longer takes place. The hornless skull of

[1] Abstracted from the *Proceedings of the Royal Society of Medicine.*

[2] The expenses incurred by these observations were defrayed partly by a grant from the Scientific Grants Committee of the British Medical Association, and partly by the Lister Institute, at the Elstree farm of which the calf to be referred to was kept for the chief part of the time.

[3] Certain of the functions of the ovary as an organ of internal secretion have been discussed by F. H. A. Marshall and W. A. Jolly (*Phil. Trans.*, Royal Society, 1905-6, p. 123). These deal with phenomena connected with pregnancy, and œstrum or "heat."

the castrated Herdwick resembles that of the ewe; the plane of the os frontis is continued backwards behind the orbits at a very obtuse angle, whilst in the intact ram the plane of the frontal behind the orbit lies almost at a right angle with the inter-orbital portion of the bone, the horn-cores arising from the upper or horizontal area. One might be tempted at first to ascribe this failure of cranial change to the absence of horns, but more probably both results are due to the same cause, seeing that the horns of the sheep do not come, like those of the rhinoceros, into the category of mere cutaneous appendages, but are provided with osseous cores. In regard to the long bones, one of the well-recognized results of castration carried out in the young animal is an increase of their length.

A more striking example of such indirect glandular influence upon the growth of bone than that furnished by the increase in length of the lower limbs after castration, is the well-known one of cretinism, where the deficiency of thyroid secretion is followed by diminished intra-cartilaginous growth of the several bones of the skeleton.

A question of theoretical interest here arises on which we may briefly digress. Is the striking shortness of the limbs in certain varieties, occurring amongst species of animals in general well proportioned, due to a thyroid deficiency which has become inherited? The most widely known example of short-limbness is the dachshund. Amongst sheep the same thing is illustrated in the ancon short-legged variety, to which Darwin refers.[1] In this case the variety arose *per saltum* as a sport, in a ram lamb. The animal was born in Massachusetts in 1791, and from this one lamb the breed was raised. As these sheep could not leap over fences it was thought that they would prove valuable, but they have been supplanted by Merinos. These sheep were remarkable for transmitting their peculiarity so truly that their original describer, Colonel Humphrys,[2] never heard of but one —questionable—case of an ancon ram and ewe not producing ancon offspring; when they are crossed with other animals their offspring, with rare exceptions, perfectly resembled either parent, no intermediate forms appearing. Amongst bovines, *the* example is the Dexter, a variety which appears to be indigenous in the South and South-west of Ireland, and of which a certain number of herds are kept in England.[3] The feature of the breed is the shortness of the

[1] "Variations of Animals and Plants under Domestication," first edition, i., p. 100.

[2] *Phil. Trans.*, London, 1813, p. 88.

[3] A full account of these cattle, and of the cretinism so frequently occurring in the calves, has been given by one of us (C. G. Seligmann) in the *Transactions of the Pathological Society*, lv., p. 1.

limbs. The head presents, like that of the dachshund, no cretinoid deviation of form. Now the noteworthy circumstance in connection with the short-limbed Dexter is the frequency with which it bears cretinous or "bull-dog" calves—a frequency far exceeding anything that obtains in ordinary breeds of oxen. In one herd, for instance, in this country in the year 1901, out of twenty-one births there were no fewer than seven cretins. This association of an apparently simple short-limbness in the adults with cretinism in the calves so strongly suggests that the former may be due to a thyroid defect (insufficient to produce the full condition of cretinism, but of a kind capable of producing one of its features) that we were led to test the effect of prolonged thyroid feeding upon the young dachshund.

As this observation has not hitherto been published we may here record it, although the result was negative. The dachshund, a female, was born on January 1, 1906. It had distemper in April, and was convalescent in May. The thyroid administration was commenced on May 10—*i.e.*, when the pup was under five months of age. The batches of tabloids used were freshly prepared (from the dispensary at St. Thomas's Hospital); each contained 3 gr. of dried sheep gland (Thyroideum siccum, British Pharmacopœia). For the first week a single tabloid was given each day, during the next week two, then three, and after the fourth week the daily dose was four. No ill-effects were at any time observed. The last four tabloids were given on May 12, 1908. The dog was thus kept under thyroid administration, with only an occasional week's intermission, for two years. No elongation of the limbs beyond what is normal to the variety has taken place during the period of growth, and the animal, which is at the present time perfectly well, presents all the marks peculiar to this variety of dog.

The Results of Oöphorectomy in the Pig.

In the Natural History Collection at South Kensington there are the skeletons of two examples of Potamochœrus, the pelves of which we have measured, with the following results:—

1364 B: Male, fully grown. Symphysis pubis synostosed; no epiphysial lines on the long bones. Pelvis: Maximum transverse diameter of inlet, 80·5 mm.; antero-posterior diameter, taken from the summit of the symphysis to the sacral promontory, 103 mm. Femur: Length taken by means of callipers from the summit of the head to the lowest point of the inferior articular extremity, 206·5 mm.; this bone was measured as an index to the size of the animal.

1363 B: Female, fully grown. Symphysis pubis synostosed; no epiphysial lines on the long bones. Pelvis: Maximum transverse diameter of inlet, 80·5 mm.; antero-posterior diameter, taken from the summit of the symphysis to the sacral promontory, 104·5 mm. Femur: Length, taken as in the preceding specimen, 182·5 mm.

The length of the femur in the female of these two specimens is 24 mm. less than in the male—*i.e.*, the animal is somewhat smaller—nevertheless, the diameters of its pelvis are equal to those of the larger male.

These measurements were sufficiently promising, as indicative of a sexual differentiation in the pelvis, to lead us to take the common domesticated pig for studying the results of oöphorectomy.

In October, 1903, three piglets were selected from the same litter; one female was kept intact, one was spayed, one male was castrated. Nothing untoward occurred after the operations, which were done by an expert "cutter," the whole of the uterus, with the ovaries, being removed through a small abdominal incision, and the testicles by an incision through each half of the scrotum. The animals were killed when full grown, November 2, 1904, December 6, 1904, January 20, 1905, and of each the pelvis and right femur were prepared by maceration. The measurements in the case of the two sows are as follow:—

SPAYED SOW.

Antero-posterior diameter of inlet of pelvis	93 mm.
Transverse diameter of pelvic inlet	65 mm.
Extreme distance between the outer borders of iliac crests ...	166 mm.
Height of pelvis from ischial tuberosity to the summit of the iliac crest	236 mm.
Length of right femur from summit of the great trochanter to lowest point of condyles	206 mm.

UNSPAYED SOW.

Antero-posterior diameter of inlet of pelvis	91 mm.
Transverse diameter of pelvic inlet	64 mm.
Extreme distance between the outer borders of iliac crests ...	163 mm.
Height of pelvis from ischial tuberosity to the summit of the iliac crest	231 mm.
Length of right femur from summit of the great trochanter to lowest point of condyles	194 mm.

It will appear that all the measurements of the pelvis of the spayed slightly exceed those of the unspayed animal. The differences in diameters of the inlet of the two pelves do not amount to more than an excess of 2 mm. on the antero-posterior and of 1 mm. on the transverse. The femur of the spayed is 12 mm. longer than that of the unspayed, showing that the animal was the larger. This reduces the

very small differences in the pelvic diameters to *nil*. In the castrated boar the antero-posterior diameter of the pelvic inlet is 85 mm.; the transverse is 65 mm.; the extreme length of the right femur is 202 mm. As one effect of castration is to increase the length of the long bones, very little can be deduced from the above measurements, as the standard of size is thus vitiated. The transverse diameter of the pelvic inlet is equal to that of either of the sows; the antero-posterior is less.

The differences brought out in the pig being so small as to be indecisive, it became imperative to make use of an animal of much larger dimensions, and one in which there is normally a pronounced difference between the pelves of the two sexes. It is probably true generally that in animals (such as the rabbit and pig) which bring forth a large number at a birth there is but little pelvic differentiation, whereas in those which bear but one or two young the size of the fœtus would, as compared with the parent, be relatively larger; and this would entail an increased capacity of the true pelvis on the part of the female.

The Results of Oöphorectomy in the Ox.

These considerations led us to test the question upon the bovine. In the British Museum (Natural History) there are the skeletons of a full-grown male and female European bison and those of a fully-grown Chillingham bull and Chillingham cow. The following measurements, which we were enabled to make, bring out a difference in the size of the pelvis in the two sexes of these cattle:—

European Bison (*Bos bonassus*).

Male (*fully grown*).

Pelvis	Transverse diameter of inlet	163 mm.
	Antero-posterior diameter in inlet	224 mm.
Femur (length taken by means of callipers)		435 mm.

Female (*fully grown*).

Pelvis	Transverse diameter of inlet	171 mm.
	Antero-posterior diameter of inlet	247 mm.
Femur (length taken by means of callipers)		396 mm.

Chillingham Oxen (*Bos taurus*).

Male (*fully grown*).

Pelvis	Transverse diameter of inlet	146 mm.
	Antero-posterior diameter of inlet	204 mm.
Femur (length taken with callipers)		351 mm.

Female (*fully grown*).

Pelvis	Transverse diameter of inlet	163 mm.
	Antero-posterior diameter of inlet	219 mm.
Femur (length taken with callipers)		330 mm.

In these four skeletons it will be found from the measurements given that in each of the females the diameters of the pelvis exceed those of the males, although the length of the femur is less :—

		Male (bison)		Female
Pelvis	Transverse diameter	163 mm.	...	171 mm.
	Antero-posterior diameter ...	224 mm.	...	247 mm.
Femur (length)		435 mm.	...	396 mm.
		Male (Chillingham)		Female
Pelvis	Transverse diameter	146 mm.	...	163 mm.
	Antero-posterior	204 mm.	...	219 mm.
Femur (length)		351 mm.	...	330 mm.

On April 27, 1906, a calf was spayed (from the flank) by Mr. F. L. Gooch, F.R.C.V.S., of Stamford, Lincolnshire. The animal was all red in colour, of the Shorthorn breed, and was exactly 7 weeks old when the operation was carried out. Recovery was uninterrupted, the external wound healing by first intention. The parts removed comprise the ovaries, together with the Fallopian tubes, and a portion of each uterine cornu 5 cm. in length; where cut across the cornu has a diameter of 0·6 cm. Each ovary is 2 cm. in the longer diameter; in the periphery of the divided surface there are a series of follicles (the largest 2 mm. in diameter), the presence of which gives a moruloid character to the free, uncut exterior.

The animal was shortly afterwards transferred to the Elstree farm of the Lister Institute, where it was kept until August 4, 1908, when it was killed, being at that date 2 years and 5 months old, and in perfect general condition. The teats were diminutive; they measured in length not more than 2 cm., and were correspondingly reduced in diameter. Each of the mammary glands was represented by a huge mass of fat. The pelvis was taken out entire, together with the several bones of the right hind limb; and the skull, with its short, slightly curved horns, was likewise kept. Before being killed the height of the animal was taken with a vertical measuring rod and sliding cross-piece furnished with a spirit-level. The height from the withers (the highest point between the shoulders) to the ground was a fraction over 12 hands (48¼ in.). Measured with a tape from the withers over the trunk and down the fore-limb, the distance was 50 in. From the withers to the root of the tail the distance, with the tape, was 51 in. The distance, with the tape, between the centres of the rounded eminences formed by the crests of the ilia was 17 in. The maceration of the pelvis was first conducted so as to leave the bones connected by their ligaments: the pelvis was allowed to dry, and was measured. The maceration was afterwards carried to completion and the bones disarticulated.

MEASUREMENTS OF THE PELVIS OF THE SPAYED ANIMAL.

Antero-posterior diameter of the inlet	19·8 cm.
Transverse diameter of the inlet...	14·7 cm.
Extreme distance between the iliac crests, taken from the outer borders	41·3 cm.
Extreme distance between the ischial tuberosities, measured from the inner side	14·3 cm.

The bones of the right hind-limb, after maceration, placed in natural apposition and in the extended position, gave a total length of 46 in. (116·8 cm.). In November, 1908, Mr. Gooch was able to supply us with the corresponding parts of a fully-grown, intact cow, aged 6 years, of the same breed—viz., Shorthorn, red. The animal was suffering from double pneumonia and pleurisy. The measurements, taken a day or so before it was killed, were: Height, just over 50 in.; length, from withers to root of tail, 50 in.; width between hips, 18 in.; the animal was just 1 in. higher over the hips than over the withers. After maceration the pelvis of the unspayed animal gave the following measurements:—

Antero-posterior diameter of the inlet	24·0 cm.
Transverse diameter of the inlet	21·5 cm.
Extreme distance between the iliac crests, taken from the outer borders	56·0 cm.
Extreme distance between the ischial tuberosities, measured from the inner side	20·1 cm.

The bones of the right hind-limb, after maceration, placed in natural apposition and in the extended position, gave a total length of 49⅝ in. (126·4 cm.). There is thus a difference in the length of the right hind-limbs, that of the spayed animal being 3⅝ in. (9·6 cm.) shorter than that of the unspayed. When the diameters of the pelvic inlet are worked out in proportion to the length of the limb the results are as follow:—

Length of limb of unspayed : Antero-posterior diameter of inlet of unspayed :
Length of limb of spayed : Antero-posterior diameter of inlet of spayed

126·4 : 24 : : 116·8 : *x*

The proportionate antero-posterior diameter in the spayed animal would be 22·2. As a matter of fact, this diameter of the pelvis in the spayed animal is only 19·8 cm.

Length of limb of unspayed : Transverse diameter of inlet of unspayed : :
Length of limb of spayed : Transverse diameter of inlet of spayed

126·4 : 21·5 : : 116·8 : *x*

The proportionate transverse diameter in the spayed animal would be 19·8 cm. As a matter of fact, this diameter of the pelvis in the spayed animal is only 14·7 cm. There is a pronounced difference in

the distance between the ischial tuberosities as measured from the inner side. Taking, again, the proportion between the limbs as a standard, the distance between the tuberosities in the spayed would be 18·5 cm., whereas it is really only 14·3 cm. And, lastly, there is a pronounced difference in the width between the iliac crests. Taking the proportion between the limbs as a standard, the distance between the crests in the spayed animal would be 51·7 cm., whilst it really is only 41·3 cm. The proportion between the antero-posterior and transverse diameters of the inlet, moreover, differs. In both, the former exceeds the latter (contrary to the case in man), but the excess in the unspayed is less than in the spayed—*i.e.*, the transverse diameter is relatively greater than the antero-posterior.

SPAYED.

Antero-posterior diameter of inlet	19·8 cm.
Transverse diameter of inlet	14·7 cm.

UNSPAYED.

Antero-posterior diameter of inlet	24·0 cm.
Transverse diameter of inlet	21·5 cm.

If the proportions obtaining in the spayed are calculated out for the unspayed, the antero-posterior diameter of the unspayed—20 cm.—would be associated with a transverse diameter of only 17·8 cm., as against the actual 21·5 cm. This observation upon the bovine demonstrates, therefore, that the growth of the pelvis is reduced as a result of double oöphorectomy when carried out at an early age.

ARMY VETERINARY SERVICE.

COLONEL L. J. BLENKINSOP, A.V.S., D.S.O., Principal Veterinary Officer, Northern Command, has been appointed in a similar capacity to Salisbury from April 18.

BY a notification received from the War Office, we are informed that an increase of five officers to the establishment has been sanctioned, as from April 19, 1910.

Clinical Articles.

CHRONIC ABSCESS IN THE MESENTERY, WITH FISTULÆ OPENING INTO THE SMALL INTESTINE; COLIC, ENTERITIS, AND DEATH.

By H. KENDALL, G.M.V.C., Melbourne.

The patient was an aged, grey, draught gelding, admitted to the hospital about 8 a.m. on September 16, 1909. The history of the case was as follows: The horse had been and was paddocked close to the stables. On the 15th he was taken into the stables and given a good feed of bran and chaff at midday, after which he was put to work. Nothing abnormal was observed till after returning to the stable, when he was found to be uneasy, would not eat his food, and soon manifested all the symptoms of ordinary colic. A drench was given by a blacksmith, the contents of which were unspecified, but judging by the appearance of the nostrils and the mouth it contained oil. The symptoms continued through the night till the next morning, when the animal was sent to the hospital.

The symptoms when admitted were temperature, 101·4° F.; pulse, normal; respirations, 16; visible membranes injected; colicy symptoms; rolling; groaning; turning head towards the flank, &c. On oscultation the bronchi were found to contain fluid, due probably to incompetent drenching.

From the history and general symptoms the diagnosis was subacute enteritis following impaction colic; also slight broncho-pneumonia from drench entering the lungs. On admittance the patient was given a drench containing spirits of ether nit.; spt. ammon. ar.; tinct. cannabis indicæ, āā ʒii. He appeared to have some difficulty in swallowing, but showed no coughing or irritation. Sood afterwards he became greatly distressed, and the breathing became laboured and abdominal, and a quantity of the drench returned through the nostrils. The exciting symptoms disappeared after a short time, but as there was no passage of fæces arecoline hybromide gr. i. was administered subcutaneously. This induced salivation and increased abdominal sound, but did not cause any expulsion of fæces. The symptoms subsided slightly, but later he began backing all round the box, while his expression became very sad-looking. The temperature remained the same all day until about 6 p.m. It then began gradually to rise, as very violent symptoms of abdominal and thoracic pain set in, the breathing became rapid, pulse

weak and greatly increased in frequency. At about 8.30 p.m. he commenced rolling violently and groaning. The temperature rose to 105·4° F., the face had a very haggard expression, the eyes sunken, ears and lower lip hanging loosely, and slight discharge from nostrils. These symptoms increased in severity till shortly before the death of the patient at about 11.30 p.m., which was preceded by a short period of semicoma.

A *post-mortem* examination was held the following morning. On opening the abdominal cavity nothing very abnormal was seen except that there was slight peritonitis in small patches; the small intestines were filled with fluid, and the contents of the great and floating colons were small and comparatively dry. On removing the intestines from the cavity a large ovoid mass, circumscribed and firm in consistency, about 6 in. diameter and about 8 in. in length, was found in the mesentery about the centre of the small intestines, to which it was firmly attached, though not occluding the lumen. On section this mass was seen to be composed of dense fibrous tissue with irregular areas of necrosis throughout. The necrotic areas were composed of dirty semi-purulent material, and communicated with the intestine by means of two irregular tortuous sinuses, the lumina of which were about the average diameter of a pencil. The walls of these sinuses and of the necrotic areas were friable and dirty brownish or black in colour. At the periphery of the fibrous growth was an egg-shaped abscess composed of thick greyish-yellow purulent material. The wall of the intestine in the vicinity of, and for a distance of 18 in. posterior to, the opening of the sinuses was thickened and in a state of acute inflammation, the mucosa showing patches of necrosis. Microscopical examination of the purulent material from the peripheral abscess showed many chains of streptococci lying amongst the pus cells, many of which contained cocci. The necrosed material from the other degenerated areas of the tumour, and from the sinuses showed, besides cocci, numerous bacilli morphologically similar to the necrosis bacillus. The lungs were extensively affected with acute broncho-pneumonia in the anterior lobes, and the inferior borders of the main lobes, while section showed the presence of fluid and ingesta in the bronchi. It was evident that as far as the lung lesions were concerned they were the direct result of maladministration of the fluid drench. The colic and enteritis were, however, due to other causes.

It seems more than probable that originally a streptococcic infection of a mesenteric gland had occurred (possibly a concomitant

of a previous attack of strangles, although the history, so far as could be ascertained, was valueless on this point). The result was the formation of the large tumour mass over the intestine. Ultimately the pus burrowed by at least two fistulæ into the small intestine, and so permitted the entrance of other bacilli such as the *B. necrophorus*. About the same time the irritation extended to the wall of the small intestine, and particularly the mucosa, inducing the colicy symptoms first observed. It is quite possible that, had a more conservative line of treatment been pursued, both as regards the primary drench, and the subsequent treatment with arecoline, the case would have at least survived longer, and perhaps recovered temporarily. At all events, as the *post-mortem* evidence showed, the case was one that could not satisfactorily respond to surgical treatment alone, but the symptoms of course did not in any way assist in the formation of a correct diagnosis.

ABDOMINAL OPERATIONS IN THE HORSE AND THE SUPPOSED RISK OF PERITONITIS.

By W. STAPLEY, M.D., M.R.C.V.S.,

Professor of Veterinary Surgery in the University of Melbourne, Australia.

The idea has long been held by veterinarians that the equine peritoneum is so liable to become fatally inflamed when opened as to be beyond the range of practical surgery. It is difficult to see why the peritoneum of the horse should be more liable to peritonitis than the human peritoneum. I am convinced, however, that from the anatomical standpoint surgery on the abdomen of the horse is difficult.

Years ago Farmer Miles entered the peritoneum under crude conditions, and his work was followed by a low percentage of fatal peritonitis. Later, Macqueen has shown that the peritoneum can be opened successfully. Hobday has by long practice refuted the belief that the horse is peculiarly liable to peritonitis. Of course, I understand that instead of writing of peritonitis I should be writing of septic infection, but as *peritonitis* is the term I, in my younger days in England, was taught to dread, I prefer to use it in these notes as an entity, simply as a mark of reverence. I have in England shot, on account of my dread of peritonitis, several "staked" horses. One was a valuable hunter at Dartford with a protruding knuckle of clean, unpunctured bowel, which I now believe should have been saved. The owner of this animal was annoyed with the unreasonableness of a man who was not game to try his best. He had every reason for his

annoyance. There has been too much gun and too much pole-axe in veterinary education of the past.

From September to December, 1909, I performed on the horse a number of abdominal operations, two of which rank as major peritoneal operations, the remainder being of minor major peritoneal operations. This work has been done under conditions not sanctioned by human surgeons, and had similar work been done on the human subject with fatal result it would have been an easy matter to get expert surgical evidence that such work justified a charge of manslaughter. The animals had to be thrown on a dusty tan bed, between two rows of ordinary horse boxes, yet in none of these cases has a general peritonitis been induced. From a clinical or practical standpoint peritonitis has been conspicuous by its absence.

Case 1.—Three-days'-old foal. Non-closure of the abdominal wall, the skin being in contact with the peritoneum. In the scrotal area a mass, looking somewhat like a cow's udder in size and shape, and chiefly composed of intestines, hung down to the hocks. The floor of the abdomen was absent practically between the umbilicus and the pelvis. At the lower border a patch of skin as large as the hand was completely devitalized, and partly gangrenous. From the xiphoid to the umbilicus a second hernia about six inches long in diameter existed, which, owing to the mass of intestines being in the scrotal hernia, was not tense. The foal was skilfully anæsthetized by Mr. Meyers, veterinary student, using the open method with a mixture of 60 per cent. ether and 40 per cent. chloroform. The area of operation was prepared with hot sterile soda solution, dried and freely swabbed with 1 to 1,000 biniodide of mercury and spirit; my hands were prepared with chlorinated lime and soda; instruments, ligatures, sutures, and gauze were boiled in soda solution. Warm, sterilized normal salt solution was used during the operation. The area of necrosed skin was at once removed, and the sac opened. It then became a difficult task to return the eventrated intestines to the abdomen proper, which, owing to much of the intestines being extra-abdominal, seemed too small for its normal content. In the difficult work I was greatly assisted by Mr. W. T. Kendall, M.R.C.V.S., the father of veterinary teaching in Australia. After the intestines were returned, the mass being retained meanwhile by means of sterilized butter-cloth, the edges of the abdominal wall were brought together by a dozen alternate sutures of silver wire and kangaroo tendon. Because of the unwieldy mass of skin about the hernial sac, and being tired of the operation (I had previously

said the ventral hernia was too large for satisfactory operation, and had only operated for demonstration purposes), I committed the error of doing slovenly surgery on this loose skin, bringing it together imperfectly with silkworm gut sutures. I did not expect the case to rally from the anæsthetic to which it had been subjected for over two hours. It did well, however, and never at any time showed anything but buoyancy of spirits and normal appetite. Of course the skin sac became septic; not, however, until the edges of the abdominal wall had united, and the peritoneum was sealed against infection. The return of the bowels to the abdomen increased the tension of the anterior hernia, and it became very prominent. Twenty days after the first operation we operated on the second, while pus was still oozing from the scrotal wound. Being apprehensive of sepsis, I was induced to use a French depilatory compound of monosulphide of sodium, quicklime, and starch in equal parts on the area to be operated upon. This removed the hair and the superficial epithelium, and we were able to get aseptic healing with the second wound, although the depilatory induced a dermatitis, which scabbed, and ultimately showed small areas of pus beneath the scabs. In this hernia the peritoneum was not opened, buried silver wire was again used, and the skin wound was brought together by subcutaneous silkworm gut suture. A row of silver stitches, therefore, extended, and still extends practically from the xiphoid to the pelvis.

The foal rapidly recovered from the effects of both operations. It developed an excellent appetite and a frolicking disposition. At the present time, three months after the first operation, all that remains to be seen externally is a thickening of the scrotum, the result of sepsis, which I attribute partly to my defective method in not closing the wound properly.

NOTE. — Peritoneum invaded in every part by my hands. No peritonitis.

CASE 2.—Draught horse, aged 2. Cryptorchid right side; umbilical hernia the size of testicle removed from top of inguinal canal; umbilical hernia closed with silver wire and silkworm gut. Uneventful recovery.

NOTE.—No peritonitis.

CASE 3.—Staked, aged cream horse. On December 2 the horse was staked on a picket fence. Result, a jagged wound 15 in. long, tearing pectoral muscles and perforating the abdomen behind the xiphoid. There was extrusion of the colon, which was perforated in three places, one wound being 6 in. long, one 3 in. long. and one

a U-shaped wound, stripping the external coats and leaving the mucosa intact. Free arterial hæmorrhage from colon, spasm of the bowel at intervals, with passage of bowel content. The case reached the hospital fully two hours after the accident. An unsuccessful attempt was made, using cocaine locally, to close the large wound in gut. Then the wound was protected with gauze (unsterilized), cotton and sheeting, and the horse cast as gently as possible—a difficult task successfully carried out by Mr. Hector Kendall, G.M.V.C., with Australian single hock tackle. The horse was chloroformed by Mr. McIndoe, student. Owing to the term being ended, my stock of supplies was incomplete. The wound was cleansed with saline solution and the gut brought further out of the wound to make sure no perforation other than those noted existed. By this time the edges of the bowel wound were œdematous. About an inch on either edge was excised and the wounds closed with No. 3 milliners' needles and boiled linen thread. Connell stitch was used as far as possible. In some places a continuous stitch was used to control the hæmorrhage; in other places Lembert's interrupted stitches were used. At the completion of the bowel work the gut appeared in good condition. It was again washed with saline and returned to the abdomen. The cramped position necessary to adopt in such an operation is to me very exhausting, and I was assisted at this stage by Mr. Lewis, student, who closed the abdominal wall with silver wire and a double drainage tube was inserted. The tags of skin whose vitality was impaired were trimmed off, and the skin wounds brought together with silkworm gut. The abdominal wall was lacerated to shreds, and had it not been for tension we should have excised a large area. The operation lasted one and a half hours, and was extremely tiring to all operators. The horse recovered from the chloroform fairly well and was put on large doses of strychnine subcutaneously. During the rest of the day a fleeting tympanites caused me to push strychnine.

December 3.—Temperature 101·5° F.; pulse 70; respirations undisturbed, bowels acting. Horse anxious for food. Muzzled and given frequent enemata of hot saline.

December 4.—Temperature and pulse normal; respiration shallow. Wound painful; rest of abdomen normal. Salines per rectum; bowels acting freely. Strychnine stopped. Drainage tube now removed (probably it would have been better surgery to have removed it twelve hours after operation). A sagging of the abdomen towards the wound suggested inaction of the recti muscles. Five p.m.,

temperature 100·6° F., pulse 56: respiration shallow. Surface wound becoming fœtid.

Dec. 5.—Temperature 101° F.; pulse 48; respirations shallow, 20 per min. Abdominal wound gangrenous. Skin shows pockets of œdema.

December 6.—Temperature 100·4° F.; pulse 43; respirations 36 and shallow. Horse weary. The wound had now lost its foul odour. Removed a slough of the elastic tissue. Blood count 5,000 leucocytes per c.mm.

December 7.—Temperature 102.4° F.; pulse 60; respirations 40, shallow. Removed the silver sutures and a quantity of necrosed abdominal wall. Colon visible for an area of circle 1½ in. in diameter. Washed out all skin pockets with 1-1000 bichloride. Cleansed bowel with hot saline. Bowels acting fairly well. Packed wound with cyanide gauze and cotton backing and applied a roller bandage, which had a perceptible effect in making the respiration less shallow.

December 8.—Wound discharging freely but looking decidedly better. Temperature 100·2° F.; pulse 46; respirations 44. Horse weary and thirsty, and becoming somewhat emaciated.

December 9.—Temperature 100·5° F.; pulse 47; respirations 40. Wound decidedly improved. The chances of a fæcal fistula forming appeared less. The visible colon has the appearance of being able to put up a successful resistance.

December 10.—Horse fed on damped oats and chaff. Good appetite. Wound doing well. Temperature 102·2° F.; pulse 48; Respirations 40.

December 11.—Appetite good. Temperature 102·2° F.; pulse 46; respirations 40.

December 12.—Temperature 102·2° F.; pulse 46; respirations 40. Horse in good spirits. Abdomen fairly full of food. Passes large stools of a fœtid character, normal in appearance, neither coated with mucus, blood nor pus. Chest percussion normal note. Accumulation bronchial breathing over both lungs. (I am forced to incidentally note that had I used my favourite anæsthetic—ether—I should have said that I had an ether pneumonia. In man it is not a rare occurrence for pneumonia to follow an injury, and if ether has been used it is invariably called an ether pneumonia.)

December 13.—To-day shows a general relapse. Temperature 105° F.; pulse 65; respirations 45. Irritability of bladder, frequent urinations, erections. Good appetite. Bronchial breathing pronounced, also a marked frimitus—not true vocal frimitus, but agreeing in time with the loud bronchial breathing.

December 14.—Temperature 101·5° F.; pulse 50; respirations 45. Horse weary. Eye very bright.

December 15.—Temperature 102·8° F.; respirations short and bad. Bronchial breathing very pronounced. Wound granulating in a normal manner—no pain around its margins.

December 16.—Temperature 102·2° F.; respiration 60. Appetite good. Œdematous swelling round right fore-arm. During day showed fleeting colic. During the dressing of wound the box door was left open and the horse went to the tan bed and rolled in pain; got up and walked back to his box.

December 17.—Temperature 103° F; respiration taxed to its utmost. Loud bronchial breathing all over chest. Percussion note slightly below normal. Horse passed a wretched night. At 10 p.m. colicky pains set in and lasted until 3 a.m.

December 18.—Temperature 102·2° F. Eye bright. Respirations can't be worse. For the first time horse refuses all food and drink. At 10 p.m. left with the idea he could not last the night.

December 19.—Found stiff and cold. *Post mortem*:—Both lungs—acute croupous pneumonia with a few purulent areas throughout. Left lung a patch of diaphragmatic adherent pleurisy. Heart—healthy, containing black and yellow clot. Wounds in bowel completely healed. Contents of bowel normal. Small abscess in abdominal wall. Nonseptic adhesions between colon and edges of parietal wound. Between liver and diaphragm many adhesions of recent origin. Professor Gilruth reports the smears from lung to show enormous numbers of streptococci in a state of purity.

Observations.—This animal died of streptococcic pneumonia, due probably to absorption from the gangrenous area of the abdominal wall. Peritonitis as such was not the cause of death. I believe that had our treatment of the abdominal wall been less conservative we should have occluded the atrium of infection. I am quite satisfied with the healing of the wounds in the colon, for from them no leakage occurred, although the bowel was at the time of *post-mortem* full of ingesta. The infection apparently started in the abdominal wall. At the time of operation this lacerated and shredded area should have been excised and sutures applied to sound tissue. Twice tiredness in myself has forced my surgical judgment and caused me to leave the abdominal wall in an unsatisfactory surgical condition. It is necessary for him who undertakes abdominal surgery in the horse to keep himself in good physical training. Kitchener issued an order in South Africa "that physical exhaustion will not be accepted as an excuse for being

found asleep on picquet duty—all men so found will be shot." He might have added that all veterinary surgeons who get tired whilst operating will fail.

Experimental Work.

Under deep anæsthesia we have endeavoured to find the surgical route into the abdomen. The horse's abdomen, owing to its anatomical formation, is difficult of approach. At present—but we hold the right to change our opinion—we believe the flank to be a bad route, and we think that in spite of weight the ventral areas offer the best approach. Especially do we believe that incisions should study the course of the nerves rather than the fibres of the muscle, and we have been unable to believe from our work that a *gridiron*, or even a muscle-splitting operation, is the operation of choice in the horse. It is more than probable that certain areas of the horse's peritoneum are more vulnerable to septic invasion than other areas. Case 3 suggests that the xiphoid area to which the colon is applied is strong in its resistance to infection. The other operations were in areas of gravitation drainage.

Our work makes it difficult for us to believe that the peritoneum of the horse is particularly liable to peritonitis. A few years back Dr. Fowler, of New York, brought forward what is known as the Fowler position. It is based upon observations on the human peritoneum establishing the fact that certain areas absorb septic products, whilst others do not. The pelvic peritoneum of man being that which is least sensitive to sepsis, he placed his patients in such a position that drainage took place to the pelvis. The principle of this theory has been acted upon by the surgical world, with, it is believed, most gratifying results. It appears to us that the area of least susceptibility of the horse's peritoneum is likely to be in the region of the colon with its myriads of germs, producing in its contiguity an immunity more or less perfect to sepsis. If that be so, the Fowler position for the horse is the standing position, and it at once obviates the disadvantage of the horse being unable to rest on its back. Be that as it may, the only part of the horse's abdomen which lends itself to drainage is the part usually described anatomically as the epigastric area. It is no part of this short paper to enter into a discussion of the vexed question of artificial drainage beyond briefly offering the advice: minimize artificial drainage as far as possible.

PARALYSIS OF THE ANTERIOR CRURAL NERVE.

By E. WALLIS HOARE, F.R.C.V.S.,
Cork.

Subject.—A strong cob, employed for general work, aged.

History.—While drawing a load of bricks, he started to kick, and managed to get the near hind leg over the shaft of the cart.

After some difficulty he was extricated and was found to be very lame.

On examination I found that the animal, while standing, kept the foot level on the ground, but when made to move, just as extension of the stifle was about to take place, he dropped on the affected side and the limb was unable to bear any weight. The action was characteristic of what is known as "dropped stifle." If an attempt was made to lift up the off hind limb, it was found that he could not bear weight on the affected limb.

Treatment consisted in the application of a blister to the region of the stifle and a run at grass for two months. The animal slowly improved and is now working in a trap. A very slight halt can be observed during extension of the stifle, but this does not interfere with his utility.

FRACTURE OF THE PELVIS AND DEATH FROM HÆMORRHAGE.

By P. R. THOMPSON, M.R.C.V.S.,
Market Harborough.

The subject was a hunter gelding, aged 8, the property of a M.F.H., and at the time of the accident was ridden by the second whip, and was the second horse. After being mounted the animal was walking downhill, and suddenly gave a jump and whipped round sharply, and then went lame on the near hind. The rider dismounted and led his horse about 3½ miles, where he got to a stable and left him there.

I was called to the case, and arrived about four hours after the accident and found the animal down, the inside of the thigh of the affected side being somewhat swollen. I managed to get the animal up and discovered a crepitus over the angle of the haunch, and on making a rectal examination I could still feel the crepitus that I first noticed, but could not ascertain that any other part of the pelvis was affected. I came home to get the slings and on my return found the animal dead, much to my surprise, and the swelling on the inside of the thigh was much bigger.

On making a *post mortem* I discovered a fracture right across the neck of the ileum, the bone being smashed into smithereens, and one edge of the fracture had severed the big arteries of the thigh, causing the hæmorrhage from which the animal died.

Just before death the animal became very violent while down, and this may account for the comminuted fracture, and possibly for the severing of the arteries as well.

CHRONIC ENTERITIS IN A MARE.

By P. R. THOMPSON, M.R.C.V.S.,
Market Harborough.

The subject a hunter mare, aged 7, was always a poor doer. She came up from grass this season, however, looking well. As soon as she was put at work she began to do badly, but was hunted for about half the present season. I was then called in to see the mare, and found that her teeth very sharp, so I rasped them. This occasioned no improvement, and the animal became somewhat jaundiced. I treated her for liver, but although the yellowness of the mucous membranes and fæces disappeared, she still did badly. After about five weeks' treatment she began to show signs of colic every now and then, the attacks becoming more and more frequent especially after a meal. I made a thorough examination of the mare but could gain very little information.

The pulse was fair in tone but a trifle slow and dull, the temperature fluctuated between 101° and 103° F. and during the attacks of colic the animal blew a good deal. This uneasiness occurred particularly immediately after feeding. After about ten days I found that the left lung was slighty involved, and after another three days the right lung also, but there was no rise of temperature at the time.

A second opinion was called in and tuberculosis was suggested by my colleague. I tested with tuberculin but got no reaction; the temperature at the time of inoculation however was 102·3° F.

The animal was fed on sloppy food, with gruel, eggs, and brandy, &c., and liquor pepsin and bismuth co. in ounce doses quarter hour after every meal.

A third opinion was then called in; this favoured very much that of the first consultant as to the probable original trouble, but at this period the pulmonary lesions were well established and masked the primary symptoms.

As the mare was wasting away I got permission to destroy her and chloroformed her, telling her owner I considered the case to be either gastritis or enteritis, and incurable.

On *post mortem* I discovered chronic inflamation of the duodenum, which in places was perfectly white and fibrous, all the muscular tissue having degenerated for about the distance of one foot. Other parts of the mucous membrane showed numerous petechiæ, and then a fibruos part again, and so on. There was double pneumonia present, but this was, undoubtedly, secondary to the enteritis.

Canine Clinical Notes.

AN UNCOMMON DENTAL FISTULA IN A DOG.

By GEO. H. WOOLDRIDGE, F.R.C.V.S., M.R.I.A.,
Professor in the Royal Veterinary College, London.

The subject of this note was a young bull-terrier, male, aged 8 months. He was brought to the College Clinique suffering from distemper. Attention was also drawn to a small discharging wound under the jaw, towards the front of the intermaxillary space and inclined to the right side. The history of this wound was to the effect that when the animal was about 4 months old (*i.e.*, about four months previously) his mother rather viciously attacked him, and bit him across the upper and lower jaws. The wounds, however, healed in about a fortnight; but several weeks later a small swelling appeared under the lower jaw and burst, liberating a purulent, blood-stained discharge. It then healed, only to swell up and burst again. This procedure was repeated several times before he was brought to our notice.

On examining the lower jaw we found a puncture that would just admit a small probe, which, when inserted, passed towards the right ramus, against which it could be felt to grate. The diagnosis arrived at was injury to the inferior maxilla, with probable sequestrum formation.

The treatment decided upon was to open up the channel and expose and scrape the bone and remove the dead portion, if it existed; and this was proceeded with under local anæsthesia induced by cocaine. A grooved director was inserted and the opening enlarged with a scalpel, and the depth of the cavity cleansed and further examined. A loose, hard substance could be felt, and was believed to be a piece

of bone. It was firmly grasped by means of a pair of artery forceps and drawn out, and proved to be not bone, but the permanent tush of that side. On referring to the mouth it was found that the right lower tush was missing, and there was no evidence of it ever having pierced the gum. The absence of this tooth had not been previously noticed. The cavity was then curetted and dressed antiseptically with chinosol solution and powdered boric acid. It healed readily, and the patient also recovered from his distemper.

Indexed B. A. I.

Abstracts and Reports.

THE NEED OF CONTROLLING AND STANDARDIZING THE MANUFACTURE OF VETERINARY TETANUS ANTITOXIN.[1]

By J. R. MOHLER, V.M.D., and ADOLPH EICHHORN, D.V.S.,

Bureau of Animal Industry, U.S.A.

Of the various biological products prepared for the cure and prevention of infective diseases in animals tetanus antitoxin has been probably the most extensively used by the veterinary profession. The good effects which were expected from the administration of this antitoxin in the prophylactic and curative treatment of tetanus have not, however, been all that could be desired, and it is surprising to learn of the divergent results which are obtained from its use by various practitioners. It is, therefore, only natural that tetanus antitoxin has not gained the favour of the veterinarian and is not used to the extent merited by such a valuable product. It has been suggested that the lack of uniform results which has followed the administration of the antitoxin is primarily due to the variation in the strength of the product, and, following numerous requests from different sources, it was decided to undertake the standardization of veterinary tetanus antitoxins prepared by different manufacturers, and also to determine whether these products are subject to any variations in strength. This work seemed particularly desirable in view of the address of Anderson before the June meeting of the American Medical Association, in which he stated that his experiments showed veterinary tetanus antitoxin to contain as low as seventeen to twenty-five antitoxin units per cubic centimetre, while similar examinations of antitoxin prepared for medical use contained from 150 units to 600 or even 700 units per cubic centimetre.

This paper is presented with the object of furnishing some concrete examples of the variation observed in tetanus antitoxin at present on the market, and to show the necessity for Federal supervision of all vaccines, serums, antitoxins, viruses, and analogous products, including mallein, tuberculin, anthrax and blackleg vaccine, and hog-cholera serum.

[1] From *Bulletin* 424 of the Bureau of Animal Industry of the United States Department of Agriculture.

Nature and Cause of Tetanus.

Tetanus is an extremely painful disease of all animals, including man, but it more particularly affects the horse, ass, and mule. It is caused by the *Bacillus tetani*. This organism differs from other disease-producing bacteria in the fact that it remains localized at the point of entrance to the body, which is usually through a wound. Here it multiplies and produces a powerful toxin which is absorbed into the circulation and, by acting on the nervous system, gives rise to the symptoms of the disease. From this it will be seen that tetanus is the type of the purely toxic disease and is not transmissible by contact unless the wound containing the germ is discharging pus or blood, as may be the case in tetanus following the operation of castration. Neither can the disease be transmitted from one animal to another by blood inoculation, because no bacilli are circulating in the blood. The bacillus is a spore former, and these spores are very resistant to antiseptics. It can live outside the animal body, and is quite prevalent in garden earth and in the neighbourhood of manure piles. It is an anærobic organism like the bacillus of blackleg, and consequently can multiply only in the absence of oxygen. Deep punctured wounds about the feet and legs are especially serious, as in wounds of this kind the bacilli are most likely to be implanted, and once they have gained access the absence of oxygen in these deep wounds makes the conditions favourable for their growth. The period of incubation varies from a few days to two weeks.

The symptoms come on gradually. There is stiffness of the gait and rigidity of the muscles, the appearance of the animal somewhat resembling a horse foundered in all four feet. The neck is rigid, the head extended, and the tail elevated and immovable. The characteristic symptom of tetanus is the protrusion of the nictitans membrane over the eye when the head is raised. This condition is due to a contraction of the muscles of the eyeball drawing it back into its socket and forcing out the nictitans membrane. There is no other disease in which this condition is present. The animal is extremely nervous, starts at the slightest sound, loses its appetite, and sweats freely. There is a spasmodic contraction of certain groups of muscles, especially those of the jaw, so that the animal is unable to open its mouth, from which the disease derives its popular name of lockjaw. The malady runs an irregular course, and death may occur in a few days, or the affection may be prolonged over two or three weeks. Spontaneous recovery is very doubtful, and the outlook is always grave. The mortality for some classes of animals has been given as almost 100 per cent., while about 75 per cent. of the cases in horses terminate fatally. Unfortunately the disease is seldom suspected until a relatively large amount of toxin has formed and begun to manifest its action in the patient's body.

Historical Summary.

Prior to the discovery of the cause of tetanus there were various theories advanced regarding the character of the disease. One of the most widely accepted views was that tetanus, since it generally develops as a result of an injury, was occasioned by the tearing or contusion of some of the peripheral nerves, and as a consequence the changes produced were conveyed to the spinal cord. On the other

hand, in the cases where no injury was associated with the disease it was thought to be of idiopathic origin.

With the discovery of the tetanus bacillus by Nicolaier in 1884, and its successful cultivation by Kitasato in 1889, the true cause of the disease, which previously had been involved in mystery, was established. Kitasato, in his investigations, also found that the organism is not present in the blood of animals dying of the affection, and, accordingly, he concluded that the fatal results were produced by an intoxication and not by the infection. In his subsequent work with this disease he, together with Von Behring, successfully worked out and published an immunizing method in 1890.

It has already been stated that in tetanus, unlike most of the other infectious diseases, the organism itself does not exert the destructive influence, only the toxins of the organisms being responsible for the serious results of the disease. The tetanus bacilli therefore produce a specific substance which has the toxic effect. Kitasato came to this conclusion in the course of his experiment when he succeeded in producing typical cases of tetanus in mice, guinea-pigs, rabbits, and other animals with the filtrate obtained by filtering tetanus cultures through porcelain filters. At the same time he found that it required only a remarkably small quantity of the toxin to produce tetanus with a fatal termination in these animals.

In the above-mentioned epoch-making work of Behring and Kitasato they attributed the established immunity to the effect of the blood serum, which rendered harmless the toxic substances produced by the tetanus bacillus. From the following described experiments they obtained results demonstrating that the serum had an enormous power of destroying the poison:—

Of a tetanus culture ten days old, which was freed from bacilli by filtration, 0·00005 c.c. was sufficient to kill a mouse in four to six days, and 0·0001 c.c. was sufficient to kill a mouse with certainty in two days. However, 5 c.c. of the serum from the tetanus-immune rabbits were mixed with 1 c.c. of this culture and the serum was allowed to act upon it for twenty-four hours. Four mice were each given 0·2 c.c. of the mixture, which contained 0·0033 c.c. of the culture, or more than 300 times the dose otherwise fatal for mice. All four of the mice remained well. The control mice, on the other hand, died in thirty-six hours after having received 0·0001 c.c. of the fluid. As a result of these and other experiments the authors drew the following conclusions:—

(1) The blood of tetanus-immune rabbits possesses the property of destroying the tetanus poison.

(2) This property is destroyed by the extra vascular blood and the cell-free serum obtained from it.

(3) This property is of such a stable nature that it is also effective in the bodies of other animals, so that we are in a position to accomplish noteworthy therapeutic results by means of the transfer of blood serum.

(4) The property of destroying the tetanus poison is absent in the blood of animals which have not been immunized against tetanus. If tetanus be given to susceptible animals, the toxin may be demonstrated in the blood and other body fluids after the death of the animals.

Tizzoni and Cattain have also established that the blood of

artificially immunized pigeons and dogs has a protective action on mice and rats against the tetanus toxin (1 to 2 drops of the dog serum made 0·5 c.c. of a toxin culture filtrate ineffective).

With these wonderful results as a foundation, the serum therapy of tetanus was established.

The practical application of this method was inaugurated after Schütz had found that horses and sheep can also be successfully immunized against tetanus, and that they produce an active immunizing serum. Horses are now exclusively used for the production of tetanus antitoxin, as large quantities of blood may be drawn from an immunized horse, which constitutes a great advantage in the manufacturing of this product for the market.

The immunizing serum is prepared according to Von Behring's method in the following way: Of 200 c.c. of a virulent bouillon culture, of which 0·75 c.c. kills a rabbit in three to four days, 80 c.c. is mixed with sufficient trichloride of iodine to make a 0·25 per cent. solution; similarly, 63 c.c. is mixed with enough to make a 0·175 per cent. solution, and 40 c.c. with sufficient to make a 0·125 per cent. solution, while the remaining 20 c.c. of the culture is left without any addition. The horse is then injected every eight days, commencing with 10 to 20 c.c. of the greatest dilution and continuing successively with the weaker attenuations of the culture. Finally the pure culture is injected, commencing with 0·5 c.c. and doubling the dose every five days, until no reaction to the culture is manifested and the blood shows the highest number of immunity units obtainable from that individual animal.

Mode of Action of Tetanus Toxin.—The highly poisonous qualities of this toxin and the fact that the organism causing the disease acts only through the generated toxin induced a greater number of investigators to carry out various investigations in connection therewith. The tetanus bacillus, it will be remembered, does not enter the general organism, but remains at the point of entrance (wounds), and only exerts its destructive effect through the action of the toxins which it eliminates. From the place of infection the toxin is conveyed principally by the nerves to the central nervous system, and here is its point of attack. The central nervous system, and especially the spinal cord, is first affected by the toxin, and all functional manifestations of the intoxication, as well as the increased reflex irritability, such as the tonic spasms of the muscles, are the result of the poisoning of the nerve-cells of the spinal cord. That the muscle itself is not directly influenced in its function by the tetanic poison is no longer doubted by pathologists.

The tetanus toxin is taken up by the peripheral nerve endings, and is then conducted by the nerves to the medullary centres, where it combines with the motor nerve cells. The poisoning of these cells then produces the tonic muscular spasms as well as the reflex irritability (Meyer and Ransom, Bruschettini, Stinzing, Von Behring, and others).

The mode of action of the tetanus toxin is explained by Ehrlich's side-chain theory in the following way: Every toxin molecule consists of a non-poisonous (haptophore) aud a poisonous (toxaphore) atom group. On the other hand, the protoplasm of the motor nerve cells consists of a vital nucleus and of numerous side chains (receptors), of which some possess a special affinity toward the haptophore atom groups of the toxin molecules. If molecules of the tetanus poison

enter the nerve cells, they become anchored to the corresponding receptors of the cell protoplasm by the haptophore atom group, and their toxophore group exerts a harmful effect on the vital nucleus of the cell. There follows either a total destruction of the cell protoplasm as a result of this combination or a stimulation of the cell protoplasm to defensive action which is manifested by the continued production and elimination of the receptors into the blood. The presence of these free receptors in the blood of immunized animals explains the antitoxic immunity, since these receptors are enabled to combine with the haptophore atom of the toxin molecule before it has had an opportunity to attack the cell of protoplasm.

The tetanus toxin, according to Ehrlich, contains at least two poisons, the tetano-lysin and the tetano-spasmin. He showed that on the relative proportions of these poisons depends whether toxin has stronger tetanic properties and a weaker hæmolytic action or vice versa.

Toxicity of Tetanus Toxin. Testing the tetanus toxin for its virulence, Kitasato found that 0·0002 c.c. of the filtrate of a tetanus culture proved fatal to a mouse, while 0·002 c.c. killed a guinea-pig weighing 560 grm. A rabbit, on the other hand, required relatively double the quantity of the mouse and almost seven times as much as the guinea-pig, since it required 0·04 c.c. of the filrate to kill a rabbit weighing 1·490 grm. Accordingly, of these three species of experimental animals the guinea-pigs are the most susceptible to tetanus toxin, mice coming next and rabbits last. Of all animals the horse is probably the most susceptible to this toxin.

The remarkable poisoning qualities of tetanus toxin can be seen from the finding of Briszer and Cohn; 0·00000005 grm. of their strongest tetanus toxin killed mice weighing 15 grm. Smaller doses, as 0·00000001 grm., caused tetanic symptoms in mice. The minimal lethal dose (MLD) of the standard tetanus toxin of the Hygienic Laboratory of the United States Public Health and Marine-Hospital Service is 0.000006 grm. for a guinea-pig weighing 350 grm.

Stability of Tetanus Toxin. Kitasato was the first to make extensive tests on the stability of the tetanus toxin, and while other investigators have since undertaken similar work with it the results they have obtained merely confirmed the findings of Kitasato.

In experimenting upon the effect of physical influences on the filtrate, Kitasato found that tetanus toxin is quite susceptible to heat. Subjected to 65° C. and above, it is totally destroyed in five minutes, while it resists 60° for fifteen minutes, but twenty minutes' exposure to this temperature destroys it. The drying of the filtrate does not necessarily destroy its virulence. However, a great deal depends upon the method of drying, as for instance, if dried in the incubator it is entirely destroyed, while if dried in the exsiccator over sulphuric acid or in the air at room temperature it retains its virulence. Exposure of the filtrate to diffused light showed a gradual diminishing effect upon its toxic action; however, it requires a long time before its virulence is entirely destroyed, as the filtrate which had been exposed for nine to ten weeks to the diffused light in the window still had toxic effects in large doses. On the other hand, the filtrate retains its virulence for a very long period if kept cold in a dark room, since it did not lose any of its virulence after three hundred days under such conditions. Exposure of the filtrate to direct sunlight proved that it

is entirely destroyed only after fifteen to eighteen hours, while shorter exposure showed a corresponding diminution of the toxic action of the filtrate. Dilutions of the filtrate with water or bouillon do not influence the virulence of the tetanus poison.

Of the chemical influences on the filtrate Kitasato found that tetanus toxin is very susceptible to the action of acids (especially mineral acids) as well as alkalies.

Practically the same results were obtained by Rosenau and Anderson regarding the stability of the tetanus toxin, and as a result of their extensive investigation with the dried toxin they found that it not only retains its virulence when kept in a cold, dark place, but loses its toxicity quite slowly when exposed to light, heat, and other influences. One sealed tube sent from Washington to Manila by mail arrived there without appreciable loss of toxicity. The stability of the dried toxin is of great advantage in the work of standardization of the antitoxins, as it assures accurate work and also simplifies and expedites the tests.

The Standardization of Tetanus Antitoxin.

European Methods.—With the establishment of the principles of immunizing against tetanus by Von Behring and Kitasato, it became necessary to adopt a method by which the potency of the antitoxin could be accurately determined. In Germany the testing of the tetanus antitoxin is carried out in accordance with the Von Behring method, which provides that normal serum shall be a serum of which 0·1 c.c. renders ineffective 0·03 grm. of normal toxin. This normal or test toxin is a dried toxin, 1 grm. of which has a virulence capable of killing 10,000,000 mice, each weighing 15 grm. The Italian method of standardizing tetanus antitoxin is based on the work of Tizzoni and Cattain. The toxin unit, according to this method, is the smallest amount of toxin which will kill a rabbit weighing 1 kilogram in from four to five days. The Tizzoni antitoxin contains 80,000 immunity units in every cubic centimetre; in other words, this amount of antitoxin will neutralize 80,000 toxin units.

The French method of standardizing tetanus antitoxin is carried out by the subcutaneous inoculation of a series of guinea-pigs with quantities of serum equal $\frac{1}{100000}$, $\frac{1}{1000000}$, $\frac{1}{10000000}$, &c., of their weight. Twenty hours later these test animals are given a single fatal dose of toxin, and the immunizing power of the serum is then considered to be $\frac{1}{100000}$ or $\frac{1}{1000000}$, &c., dependent upon whether the animal receiving this proportional weight of serum has survived the toxin which proved fatal to the control guinea-pig.

The American Method of Standardization.—The method of standardization which was carried out in connection with the testing of the veterinary antitoxins as reported in this paper followed the exact lines of the method known as the American method, which has been adopted officially under the biological product act of July 1, 1902.

This method, which is the result of several years' work on this subject in the Hygienic Laboratory, is a highly creditable achievement by Rosenau and Anderson,[1] as it not only simplifies the standardiza-

[1] Rosenau and Anderson. The standardization of tetanus antitoxin. *Bulletin* 43, Hygienic Laboratory, United States Public Health and Marine Hospital Service. Washington, 1908.

tion of this valuable serum, but is also perfectly reliable in its accuracy. The method was also unanimously adopted by the Society of American bacteriologists subsequent to the report made by a special committee.

In this method the immunity unit for measuring the strength of tetanus antitoxin is fixed so that it shall be ten times the least quantity of antitoxin serum necessary to save the life of a 350-grm. guinea-pig for ninety-six hours against the official test dose of a standard toxin furnished by the Hygienic Laboratory of the Public Health and Marine Hospital Service.

Thus it is required from the manufacturers of the "human" tetanus antitoxin to state the number of units their products contain, which not only insures serum of reliable strength, but also establishes a uniformity among the producers of tetanus antitoxin in America. On the other hand, the antitoxins destined for "veterinary" use are still under no control whatsoever; there is no uniformity in the method of standardization, and the potency of the product is absolutely left to the honesty of the manufacturer. This state of affairs, of course, does not assure the veterinarian of any uniformity of the product even of the same manufacturer, still less of different manufacturers. Only one of the different veterinary tetanus antitoxins on the market states the number of American units the immunizing or curative dose contains. The others fail to make any declaration, and in one case the number of units is given in the hundred thousands, whereas the human antitoxin of the same firm of course complies with the requirements of the law, stating definitely the number of American units that particular antitoxin contains. These conditions alone should suffice to point out the necessity for supervision of this veterinary product and the establishment of uniformity among the producers of veterinary antitoxin. It is very essential that the veterinarian should have some assurance of the strength of the antitoxin upon which his standing as a professional man may depend.

Examination of Commercial Veterinary Antitoxins.

The samples of tetanus antitoxin (veterinary) which were standardized in connection with this work were obtained from various drug stores in Chicago, New York, and Washington. They were kept at a temperature from 50° to 60° C. until the tests were made, care being taken not to expose them to any condition which might affect the potency of the antitoxin.

The toxin was obtained from the Hygienic Laboratory of the Public Health aud Marine Hospital Service through the courtesy of Surgeon-General Walter Wyman, and represented the dried standard toxin which is used in the standardization of tetanus antitoxin in the Hygienic Laboratory and furnished to the manufacturers of antitoxin. In the determination of the value of the antitoxin the L + dose is the test dose of the toxin. The L + dose is the smallest quantity of tetanus toxin that will neutralize one-tenth of an immunity unit, *plus* a quantity of toxin sufficient to kill an animal in just four days. The L + dose of the toxin which was used in these tests contained 100 minimal lethal doses (MLD) for a 350-grm. guinea-pig. The toxin was kept in a dark, cold place, and during the course of these

tests its virulence was controlled by repeated tests with an antitoxin of known value.

The guinea-pigs used in connection with this standardization were carefully selected, being vigorously healthy animals of from 350 to 370 grm. in weight. The glassware employed in the work was selected according to the recommendation of Rosenau, and was not used for any other purpose. The mixing cylinders and mixing flasks were the kind designed by Rosenau. Ehrlich's delivery pipettes, graduated into hundredths, as well as special delivery and capacity pipettes, were used. The syringes employed were a modification of the old Koch syringe, in which the barrel tapers gradually to the needle, so that the last drop runs out readily.

The necessary measures against bacterial contamination were taken during the execution of the work. All the glassware was first rendered chemically clean and then sterilized for one hour at 120° C. All the various steps in the procedure were followed carefully in order that the results would be accurate and reliable. The method of the standardization was executed in every particular as described by Rosenau and Anderson in *Bulletin* 43 of the Hygienic Laboratory.

The number of immunity units contained in a cubic centimetre of serum is determined by the quantity of antitoxin which saved the life of a guinea-pig for ninety-six hours against the official test dose of the toxin. This quantity of antitoxin represents one-tenth of an immunity unit. For instance, if a guinea-pig receiving 0·0015 c.c. of the antitoxin with the official test dose of toxin is saved for ninety-six hours, this quantity of antitoxin contains one-tenth of an immunity unit. Accordingly, the following equation is represented: 0·0015 : 0·1 : : 1 : x, which indicates that 1 c.c. of the serum contains sixty-six units.

Following are given the results of the standardization obtained from the tests of the various samples of veterinary tetanus antitoxin examined in this laboratory[1]:—

No. 1.—H. K. Mulford Company's tetanus antitoxin serum for veterinary use. Immunizing dose, labelled to contain 500,000 units. To be exchanged after May 1, 1910. Laboratory No. 2,960. Syringe contained 7·6 c.c. On testing, the number of antitoxin units per cubic centimetre was 81, and the total number in syringe, 615.

No. 2.—Parke, Davis and Co.'s antitetanic serum (veterinary). To be exchanged after November 24, 1910. Unit value not stated. Syringe contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 125, and the total number in syringe 1,250.

No. 3.—H. K. Mulford Company's tetanus antitoxin serum for veterinary use. Immunizing dose, labelled to contain 500,000 units. To be exchanged May 15, 1910. Laboratory No. 2,960. Syringe contained 7·5 c.c. On testing, the number of antitoxin units per cubic centimetre was 81, and the total number in syringe 607.

No. 4.—Pasteur Laboratories, Paris, France, antitetanic serum for veterinary use. Unit value not stated. Bottle contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 100, and the total number in bottle 1,000.

[1] In the original article tables are included giving full details of the experiments conducted in these tests.

No. 5.—Lederle Antitoxin Laboratories, tetanus antitoxin (veterinary). Immunizing dose, labelled to contain 1,500 units. Laboratory No. 19A. To be exchanged June 21, 1910. Syringe contained 10·4 c.c. On testing, the number of antitoxin units per cubic centimetre was 154, and the total number in syringe 1,601.

No. 6.—Parke, Davis and Co.'s antitetanic serum (veterinary). To be exchanged after December 8, 1910. Unit value not stated. Syringe contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 125, and the total number in syringe 1,250.

No. 7.—Parke, Davis and Co.'s antitetanic serum (veterinary). To be exchanged after December 8, 1910. Unit value not stated. Syringe contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 125, and the total number in syringe 1,250.

No. 8.—Parke, Davis and Co.'s antitetanic serum (veterinary). To be exchanged after January 14, 1911. Unit value not stated. Syringe contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 100, and the total number in syringe 1,000.

No. 9.—H. K. Mulford Company's tetanus antitoxin serum for veterinary use. Immunizing dose, labelled to contain 500,000 units. To be exchanged after May 15, 1910. Laboratory No. 2,971. Syringe contained 8 c.c. On testing, the number of antitoxin units per cubic centimetre was 59, and the total number in syringe 472.

No. 10.—Pasteur Laboratories, Paris, France, antitetanic serum for veterinary use. Unit value not stated. Bottle contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 111, and the total number in bottle 1,110.

No. 11.—Pasteur Laboratories, Paris, France, antitetanic serum for veterinary use. Unit value not stated. Bottle contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 153, and the total number in bottle 1,530.

No. 12.—H. K. Mulford Company's tetanus antitoxin serum for veterinary use. Immunizing dose, labelled to contain 500,000 units. To be exchanged after May 1, 1910. Laboratory No. 2,960. Syringe contained 7·5 c.c. On testing, the number of antitoxin units per cubic centimetre was 81, and the total number in syringe 607.

No. 13.—Lederle Antitoxin Laboratories, tetanus antitoxin (veterinary). Immunizing dose, labelled to contain 1,500 units. Laboratory No. 19A. To be exchanged June 21, 1910. Syringe contained 8·5 c.c. On testing, the number of antitoxin units per cubic centimetre was 232, and the total number in syringe 1,972.

No. 14.—Pasteur Laboratories, Paris, France, antitetanic serum for veterinary use. Unit value not stated. Bottle contained 10 c.c. On testing, the number of antitoxin units per cubic centimetre was 153, and the total number in bottle 1,530.

The results of these tests clearly demonstrate the variations in the potency of veterinary tetanus antitoxins at present on the market. While the preparations of the individual manufacturers do not show such marked differences in strength, yet they do not uniformly contain a sufficient number of units.

In accordance with the (United States) law of 1902, the manufacturers of human antitoxins are required to state on the labels of the packages the number of units that the particular antitoxin possesses. Should it be found on investigation that the antitoxin does not come

to within 10 per cent. of the strength stated on the labels, the manufacturer is immediately required to recall from the market all that particular antitoxin. Thus, if a physician intends to use the antitoxin either for immunizing purposes or as a curative agent, he is accurately guided in the dose by the statement on the label. It does not matter who the manufacturer of the antitoxin is. This latter fact is in itself also of great importance, as frequently the drug stores carry antitoxins of only one or two manufacturers.

On the other hand, the veterinarian has not always the good fortune of knowing the number of units that an antitoxin which he purchases contains. Only one of the manufacturers states on the label the number of American units contained in his veterinary antitoxin. One other manufacturer still uses for his veterinary antitoxin a standardization—other than American—by which he can label his product in the hundred thousands, yet his antitoxin for the human gives the units in the American standard. Why should two different standards be maintained, one for the human and the other for veterinary antitoxin?

The veterinary tetanus antitoxins are marketed in immunizing doses and curative doses. The immunizing dose is supposed to contain 1,500 American units. The volumetric quantity of this dose was found to be accurately 10 c.c. in the Parke, Davis and Co. and the Pasteur product, while the Lederle serum contained from 8·5 c.c. to 10·4 c.c., and the Mulford syringe from 7·5 to 8 c.c. Now, should it be desired to administer to a horse an immunizing dose of the antitoxin, it can readily be seen from the results obtained in these tests, as indicated by the tables, that while the immunizing dose of some of the serums [contains the desired 1,500 units, others, on the other hand, possess less than one-third of that strength. For instance, according to the test shown by No. 9, the syringe contained 8 c.c. of serum, with fifty-nine units per cubic centimetre; thus the immunizing dose in this case represents only 472 units, and of course the curative dose is correspondingly low. This alone is sufficient to indicate the urgent necessity for some uniformity in standardizing the veterinary antitoxins, and also for Federal legislation by which they could be subjected to a periodical control with reference to their potency.

Under the present conditions there is the constant uncertainty regarding the strength of the serum, as the veterinarian has no assurance whatever of its potency, and is solely dependent on the reliability of the manufacturer.

Conclusions.

(1) The veterinary tetanus antitoxins prepared by the different manufacturers have not a uniform potency, and the variation amounts in some instances to about two-thirds less than the strength which it should possess.

(2) In order to insure a uniform strength, the manufacturers of veterinary tetanus antitoxins should be required to use the American standard, and to state on the label the number of American units the dose contains, as is required for human tetanus antitoxin.

(3) The immunizing dose for a horse should contain at least 1,500 immunity units of the standard established by the United States Public Health and Marine-Hospital Service.

(4) It is seen that the veterinary tetanus antitoxins vary extravagantly in the unit strength, and some are comparatively weak in antitoxic potency, which shows the necessity for the same supervision by the United States Department of Agriculture over biological products used in veterinary medicine as is now exercised by the United States Public Health and Marine-Hospital Service over similar products used in human medicine.

(5) The request for such supervision should have the endorsement of the veterinarians and live-stock interests of this country.

COLONIAL VETERINARY SCIENCE.[1]

By Dr. ARNOLD THEILER, C.M.G.,

Government Veterinary Bacteriologist of the Transvaal.

When your Principal, Professor Mettam, wrote to me some months ago in South Africa, knowing of my forthcoming visit to Europe, and asked me to give you an address on the subject of Colonial Veterinary Science on this occasion of the opening of the new term of your College, I at once made up my mind to avail myself of this kind invitation, should time permit me to do so, which I am happy to say it did.

There were several reasons why I was pleased to accept his invitation so readily. Firstly, I wished to see the country from which several of my South African friends have hailed, men both in and out of the profession, who have proved to be such excellent colonists. Secondly, to make the acquaintance of men whose names were familiar to me through the good work they had done in the past. Thirdly, because it gave me the opportunity for the first time in the British Isles, to lay before a scientific and appreciative audience an *exposé* of a special branch of our science, namely, the Colonial Veterinary Science, and, by enabling me to draw attention to its importance, of demonstrating the necessity of giving it more attention at the veterinary colleges of the home country.

In speaking of Colonial Veterinary Science I do not mean a science exclusively peculiar to the colonies. Science is always the same, whenever and wherever it may have to be made use of; I apply the term rather to certain peculiarities of stock diseases met with in the colonies, and to the conditions under which they have to be dealt with, conditions which to a great extent differ from those found at home, and vary much according to the state of civilization of the different colonies. In this respect the colonies of Great Britain offer a multitude of variations; but the basis of all colonial enterprise is the development of a colony's own resources. This is a principle which, in the past, has invariably been applied by the British people in the administration of their over-sea possessions, and this is the secret of success. One of the resources—and in most colonies the main one—is the breeding and rearing of live stock. In most of them this forms the main occupation of the farming population. Stock are reared for

[1] An address delivered on the occasion of the opening of the new term of the Royal Veterinary College, Dublin, October, 1909, Sir C. J. Nixon, Bart., in the chair.

the purpose of providing working animals and for the production of milk, butter, and cheese, both for home consumption and for export, sheep and goats for their wool and mohair, almost exclusively for export, and without exception, in all colonies domesticated animals, whether horses, oxen, camels, or elephants, are made use of for transport, and form in this respect the first medium for the development of a country. Yet when we look back into the early history of some of the colonies, or when we look at the existing conditions in others, especially in the tropical African possessions, we find that owing to plagues and diseases of stock colonial enterprise severely suffers.

On the other hand, when we look at the progress made in the stamping out of diseases by State regulations in Europe, more especially in the British Isles, and particularly in this country, we are struck by the fact that the experience gained here did not induce the administrative authorities to proceed similarly in the colonies. It is true enough that the conditions are different, and the diseases to a great extent also differ. This is, however, not an excuse to leave things in abeyance or to consider the natural course of events inevitable, when such a vital interest as the speedy development of a country is at stake. It is further true that in the past Colonial Veterinary Science stood helpless at the approach of a new plague, and this will to some extent still be so when a new one arrives. This was due to the want of proper knowledge, which could not and cannot be gained without experience, research, and experiments. The duty of a colonizing government is, therefore, to offer such facilities as will tend to the organization and maintenance of institutions where these unknown and new diseases can be studied. The knowledge thus gained forms a base of all prophylactic measures undertaken either individually by owners of stock, transport riders and farmers, or by the administrative authorities through their organized veterinary staffs. In this respect South Africa, and especially the Transvaal, offers an example worthy of emulation by the home and other Colonial Governments, inasmuch as by inaugurating a veterinary department with an efficient staff, it has maintained and encouraged scientific research, and finally has built a modernly equipped laboratory at an expense of about £80,000, exclusively for the study of animals' diseases, and towards the maintenance of which an annual contribution of about £30,000 is granted.

All prophylactic measures against diseases have the one ultimate object in view, namely, eradication; to effect this a thorough knowledge of etiology is necessary, which can only be gained by research. There are plagues which can be successfully dealt with when they are made the subject of legislation and placed under proper control, and this is possible even in a colony where a mixed community of civilized and non-civilized people live, as has been repeatedly proved by the experience in South Africa.

For the purpose of making you more fully acquainted with the plagues and diseases existing in the colonies, I wish to dwell in the first instance on Africa, which in this study is closely related to India, or perhaps it would be better to say Asia, and I shall take the etiological moment as guide for further deliberations. We may distinguish two great groups of plagues or diseases, namely, directly contagious

and indirectly contagious ones. Under the term "directly contagious" we understand a disease for whose propagation the presence of sick animals is required, or of their products bearing the causal agent of the disease in the shape of micro-organisms commonly called virus. It will be useful to introduce an expression which is frequently used in the diseases peculiar to the Tropics, namely, the term "reservoir of the virus," meaning an animal carrying the infection. When applied to the directly contagious diseases the sick animal itself is the reservoir of the virus, the source of all contamination. Speaking now more particularly from a South African point of view, we do not know of any directly contagious disease specific for that country which has not occurred or is not still present in other parts of the world. Indeed, all directly contagious plagues were at one time or another imported into South Africa, and they are all more or less cosmopolitan. We have to consider rinderpest, pleuro-pneumonia, glanders, epizootic lymphangitis, tuberculosis, scab, anthrax, and quarter evil. The etiology of these plagues has principally been worked out in Europe, with the exception of rinderpest, the scientific study of which on modern lines may be claimed by South Africa. But it is not my intention to repeat here the micro-biology of the diseases mentioned. This must be familiar with the practitioner and the older student, and the younger generation will hear about it in due time ; it is rather my intention to show how they were and how they are still dealt with in the African sub-continent.

In this group of diseases the source of all infection, the reservoir of the virus, is the sick animal. Its removal and destruction is, therefore, the main object. To this principle we all agree, both here and in Africa. In the means to achieve it we differ.

Some years ago rinderpest swept down broadcast over the whole sub-continent, killing thousands and hundreds of thousands of cattle in its progress ; then, after the checking of its advance had proved futile, the immediate idea was the saving of the greatest number possible of the cattle that were exposed to infection. The farmers tried to save as much stock as was possible under the existing conditions, and were quite willing to bear a share of loss. This idea of saving whatever you can by means of inoculation or curative treatment does not apply to rinderpest only, but to any disease which comes suddenly and causes a great mortality, and this idea is not at all a peculiarity of the South African farmer, it is characteristic of almost any farmer wherever he is ; it was, however, particularly pronounced in South Africa, where there had been no previous history of State-aided combatting of diseases, and the farmers had to rely on their own individual efforts and resources. But even now the old principle of saving the greatest number of animals either infected or exposed to infection is still the primary object of all action, and is adhered to wherever possible ; indeed, it comes simultaneously into consideration with the ultimate object of the total eradication of a plague. Thus two objects have sometimes to be combined, one which seems to defeat the other. The following notes may illustrate what is meant :—

In rinderpest, for instance, the destruction of the virus is essential ; we achieve this either by killing the sick and susceptible animal or by rendering the latter immune ; the former method is the old one of

Europe, where probably even now it would be the only right thing to do; the latter is our method. You will be aware that two forms of immunity are distinguished, active and passive; the active immunity is obtained by actually passing an animal through a mild attack of the disease, from which it recovers. In rinderpest this is achieved by means of the injection of bile, or the simultaneous inoculation of protective serum and virus; the passive one renders the animal temporarily unfit for any infection; it is obtained by the injection of protective serum alone. The former usually lasts as long as immunity acquired under natural conditions; the latter of comparatively short period.

By rendering an animal actively immune it temporarily becomes a reservoir of virus, and thus creates and maintains a new source of infection. Thus, by using this method as a means of combatting a plague in the first instance, the creation of a large source of infection is caused, and this may be responsible for the further spread of the disease. On the other hand, the injection of serum alone does not spread disease; the protection may, however, not last long enough to outlive the period of infection. The application of either method, or a combination of both, depends entirely upon the existing conditions and on the immediate object in view; they are usually combined, and the success in stamping out the disease, and at the same time saving the greatest number of animals, depends largely on the men who have to judge which method should be adopted in each case, and what other precautions should be taken.

We also make use of active immunization in the stamping out of pleuro-pneumonia; this method is totally condemned in Europe, and I may say quite rightly. Experience in Europe has proved that not only the sick animal, but also the apparently recovered animal—in other words the chronically sick animal—acts as reservoir of the virus. For a successful stamping out of the disease its destruction is, therefore, essential. In Africa, we recommend the slaughter of every animal, but we have the experience, especially in the Transvaal, that the "lunger" does not play such an important *rôle* in the propagation of the disease as is accepted in Europe, otherwise we would not have been able to deal so successfully with this disease as we have done in the past. Here, also, our immediate object is to save the greatest number of animals by rendering them immune, which, of necessity, must stop all further propagation of the disease once complete immunity is obtained. The method consists in the subcutaneous inoculation of virus in the tail, either in the form of the pleuritic exudate or the pure culture. Gentlemen, it will perhaps surprise you to hear that in the Transvaal, in the way indicated, we have been able to clear the country of all rinderpest, and of all pleuro-pneumonia. The former disease has completely disappeared from South Africa; it is still lurking in the far north of Africa. The latter one is still present in some colonies, and is occasionally imported into the Transvaal. Its final eradication from South Africa is in view, even from native territories, where close supervision is a very difficult matter.

With regard to glanders, we make use of the approved method of Europe by tracing the infection to its source, ascertaining the affected animals, principally by means of Mallein, and destroying the reservoir of the virus. In this respect Mallein has rendered us immense assist-

ance, and its utility can in no way be demonstrated better than by informing you of the fact that, for instance, in Rhodesia, glanders has completely disappeared, after all animals reacting to the test were killed, and now only such animals are allowed to be imported as have passed the test and are found free from infection. Progress is, however, general in South Africa, and the time is not far off when a similar state of affairs in other Colonies can rightly be expected.

The principle of destroying the infected animal is also largely applied in the combatting of epizootic lymphangitis, although we allow the treatment of cases which are not badly affected.

Tuberculosis is not yet met with amongst the native cattle of Africa; lately, however, it has made its appearance amongst pure-bred and cross-bred animals of imported stock. We endeavour to prevent its spread by the destruction of infected animals and the control of importation. Here, also, we make use of the approved method of Europe, namely, tuberculin, but we are looking forward with equal eagerness as yourselves to a better method. This the more so, as the judging of thermal reactions in animals of tropical and sub-tropical countries, both after Mallein and tuberculin injections, may form a source of grave errors, sometimes due to the inconsistency of the normal temperatures of the animals, caused by unsuitable surroundings and external conditions. Accordingly, contingencies have to be taken into consideration which are negligible in this country. For instance, the use of tuberculin in tracing the disease in the half-wild cattle of Madagascar, where, in the southern part, the disease is prevalent, is quite hopeless.

In anthrax and quarter evil the approved methods of Europe are also successfully made use of, and for the protection of the exposed animals recourse to vaccinatian is largely taken. The destruction of the infected cadaver is, however, not always an easy matter, and there is still room for devising a method of disposing of cadavers where burning or burial is not possible, as is so often the case in the dry parts of Africa. Great assistance in saving the largest number of exposed animals is found in the vastness of the African veld. Herds can be moved out of the infection, so that further mortality stops. Luckily anthrax plays a relatively small *rôle* as a devastating disease. Quarter evil, however, is fairly prevalent, but it is successfully dealt with by inoculation, a method fully approved of by our farmers.

Scab is the sore point of the African veterinary surgeon, and we are a long way from arriving at the happy position of our sister colonies, Australia and New Zealand, where the disease has completely disappeared. It is not for the want of proper knowledge how to combat it; the colonies mentioned have given the example how it can be done; it is rather due to the fact that the local conditions and the custom of shepherding and trekking into summer and winter veld makes a complete control somewhat difficult. However, in the Transvaal there exists a great wish to clear the country of scab at all costs, and for this purpose large sums of money are spent.

The second group of diseases with which we have to deal in Africa is that of indirectly contagious ones, where an animal can only become infected by means of an intermediate host, the host being either a tick or an insect. These are the diseases which are commonly called the tropical ones, and although they are not exclusively typical for the

Tropics, they are nevertheless the most prevalent ones, which is probably due to the fact that in warm climates the transmitting hosts find the best conditions for their existence. All these tropical diseases are, without exception, diseases of the blood, the micro-organisms either attacking the corpuscles, such as the piroplasms, or living in the blood-stream, such as trypanosomes or spirochætes. In this group have also to be included some very important maladies caused by invisible or ultra-visible and filterable micro-organisms.

Let us consider, first of all, the tick-borne diseases. To these belong redwater, caused by *P. bigeminum;* an infection due to *P. mutans;* the formidable East Coast fever, which has caused so much damage in South Africa, due to *P. parvum;* the biliary fever of horses, due to *P. equi;* the biliary fever of dogs, due to *P. canis;* and the spirochætosis and the heartwater of ruminants, this latter caused by an ultra-visible organism. From a prophylactic point of view an important fact comes into consideration, namely, that an animal, after recovery from the disease to which it has become immune, can act as a reservoir of the virus, and as a source of constant infection from which the transmitting host, the tick, obtains the virus, which it inoculates in turn into susceptible animals. This fact is the cause of the permanent infection of the African veld with some of the tick diseases, and the reason why it is so difficult to keep the susceptible imported animal alive. The diseases in which the immune animal acts as a reservoir of the virus are inoculable with the blood of such animals, with the exception of heartwater, of which only the sick blood, and of East Coast fever, of which neither the sick nor the immune blood is infective. In order to understand the *rôle* of the tick as a carrier of the infection the knowledge of its life-history is important.

In the biology of a tick we distinguish the adult stage, in which both sexes are met with. The female lays an enormous number of eggs, usually amounting to several thousand; the eggs hatch into larvæ, these engorge and moult into nymphæ, these again engorge and moult into adults. Some of the ticks require one host only for the completion of their life-cycle, as, for instance, the blue tick, *Boöphilus decoloratus;* others require two hosts, such as the red tick, *R. evertsi*, which changes from larva to nympha on the animal, and from nympha to adult off the animal; a third group requires three hosts, all three stages undergoing their moults underground; to this belongs the group of the brown tick. According to these characteristics, the infection is transmitted either through the larva, having been taken up by the adult female and passed through the egg. Such is the case in *P. bigeminum* and *Spirochæta Theileri*, which are both transmitted by blue ticks. *P. equi*, the cause of equine biliary fever, is transmitted by the adult stage of the red tick, after it has become infected in the nymphal stage; *P. mutans* and *P. bigeminum* may be transmitted by the same stage of this tick. *P. parvum*, the cause of East Coast fever, is transmitted by the brown ticks (*Rhipicephalus appendiculatus, simus, capensis*, and *nitens*) in the nymphal or adult stage, after the infection has been obtained in a previous stage. In this disease it has been proved beyond doubt that the infection does not pass through the egg. Heartwater is transmitted by the bont tick, *Amblyomma hebræum*, in nymphal and adult stages. *P. canis*, the cause of canine biliary fever, is transmitted by the adult stage of the tick after it has been taken up by the

adult stage of the previous generation; the intervening larvæ and nymphæ, although infective, are not able to communicate the disease.

It is of the utmost importance to know the exact length of time the various ticks require for their hatching and moulting, and how long the life of the various stages lasts, because on this knowledge depends the possibility of extirpating them by starvation, and accordingly, at the same time, of the disease also. For instance, a successful interference in a case of East Coast fever lies in the fact that the responsible tick dies out within about fourteen months, after which time it is quite safe to restock an infected farm. Based on the knowledge of the length of the various stages the brown ticks require for their moulting, it is possible to undertake a successful removal of cattle exposed to infection by passing them through into a non-infected area, keeping them there during a certain period, when all the infected ticks will drop, and moving them out on to fresh ground before these ticks have moulted and have become ready to re-attach themselves. These intervals have been so timed that during the period of the first stage of the movement all infected animals are detected, in the second stage the clean ones are out of the infection, and by fencing the infected farm a guarantee is given against the reintroduction of cattle, so that the infection must of necessity die out.

Diseases in which the immune animal acts as a reservoir would be dealt with in a similar way, and the Americans have applied these principles towards the successful eradication of Texas fever. Under African conditions, however, a procedure for the eradication of diseases, the reservoir of which is the immune animal, is not yet possible, and at present not even advisable. With but a few exceptions the whole country is infected with piroplasmoses, and attempts to eradicate ticks would be futile. It is therefore advisable to make use of the existing immunity obtained from inoculation for the protection of susceptible animals. This can be done in redwater and equine piroplasmosis; the blood of an immune animal acts as a vaccine, and although this method is quite successful for African-born susceptible animals, it is not yet perfected for imported ones. It seems to me that the cause of the failure lies to a great extent in the fact that in the inoculation of blood of an immune animal, usually not only the piroplasm under consideration is transmitted, but also other inoculable and deadly diseases. One of these we are actually studying at present, and here indeed is room for more research. Country-born animals do not suffer much from these piroplasmoses; calves and foals as a rule easily recover, and remain immune for the whole of their lives through constant exposure to tick infection; it is the imported animal that dies, and herein lies the difficulty of improving the native breed of stock in the various African colonies.

To the sub-group of insect-borne diseases belong those transmitted by flies; one genus in particular, the *Glossina*, comes principally into consideration, and the diseases transmitted are due to one genus of blood parasite, namely, trypanosomes. This statement, however, only holds good for Africa; in other parts of the world *Glossinæ* are not known, yet there are trypanozoonoses, as these diseases are called. There are quite a number of species of trypanosomes described in Africa, of which the most important are *T. Brucei*, the cause of nagana; *T. dimorphon*, the cause of perhaps the most widely spread

fly disease in Africa, and some more which are not yet sufficiently determined. What is known concerning the maintenance of the infection in the case of the piroplasmoses applies also to the trypanozoonoses. The animal which has recovered from the disease and is considered immune may act as a reservoir of the virus. In the case of nagana—game principally comes into consideration as such—the fly acts as a transmitting agent from the reservoir to susceptible animals, and at one time it was thought that this transmission was purely a mechanical one, the fly playing merely the *rôle* of a simple inoculator; but for protozoological reasons and in analogy with the mosquito-carried malaria of man and the tick diseases, it began to be surmised that within the fly a certain development of the trypanosomes takes place. This has now lately been proved to be the case; it was found by Professor Kleine, and it has been supported by Bruce, that an infection can be carried by a fly, not only in a mechanical way when the trypanosomes appear after an incubation time following the date of biting, which corresponds to a simple inoculation, but after a much longer period, thus indicating that in the meantime the trypanosome develops in the body of the fly before the bite was infective.

There are several species of tsetse-fly, and the habits of all of them are not the same or yet fully known. Accidental experience has shown that in the case of some fly diseases man could successfully interfere; for instance, in South Africa the disappearance of nagana was subsequent to the destruction of game, and in this way rinderpest, which was the cause of the decimation of the larger species of game, was at the same time the cause of the disappearance of the fly diseases. It must, however, be understood that this does not hold good for all trypanosomes, and probably not for all species of flies. Fly life is to a great extent dependent on the bush, and the destruction of the bush is followed by the disappearance of the fly. But this remedy is only feasible to a limited extent, and therefore for a rational and effective prevention of trypanozoonoses a great field of research is still open.

Other insect-borne diseases may be summed up in the South African horse sickness and catarrhal fever of sheep, or, as it is commonly known, blue-tongue. For epidemiological reasons night insects must be considered as carriers, and therefore mosquitoes are suspected. Experimentally it has not yet been proved which species is responsible, a work which has yet to be undertaken. The fact has been demonstrated, however, that the exclusion of all insect life by means of insect-proof stables affords protection to the animals. These two diseases, horse sickness and blue-tongue, are seasonal diseases, depending to a great extent on the annual rainfall, and such tedlurical conditions as are favourable for the development of insect life. We only know as yet that with the blood of recovered animals we cannot transmit the disease artificially, and on epidemiological grounds we must accept the theory that some reservoir exists, which is likely to prove to be some other species of animal. It must be the work of further investigations to find this reservoir, and should it be found it will then perhaps be an easy matter to eradicate it, and with it the disease. In the meantime use of other means has to be made to combat the diseases, or at least to save the greatest number of the animals exposed to infection. This is done by making use of the immunity resulting after recovery from the disease. We have intro-

duced a sero-vaccination which protects mules against horse-sickness to a great extent, but we have not yet been able to save the same number of horses. Also against blue-tongue in sheep we have introduced a successful vaccination which protects highly, and which permits of re-stocking farms where on account of blue-tongue they had become uninhabitable for sheep. In the case of horse-sickness in horses various degrees of susceptibility and of immunity offer a very complex problem which can only be solved by numerous experiments.

A fourth group of diseases is due to intestinal parasites, and in this respect sheep sometimes suffer so severely that in certain parts successful sheep farming is impossible. Here is a further opening for research, especially into the biology of these parasites for the purpose of successfully interfering from a prophylactic point of view.

Finally, it is incumbent on me to inform you what is not generally well known in Europe, that there are diseases met with in Africa amongst stock of all kinds which are not yet properly known or studied, and against which we are as yet helpless in advising definite means of treatment. I need not mention the names, it is sufficient to state our present helplessness and the necessity for further research, demonstrating to you the possibility of solving great problems.

From my notes on Colonial Veterinary Science, I hope to have been able to show you the importance of the subject, both from the economic as well as the scientific point of view, and therefore the necessity of giving it as much attention as it deserves in the veterinary colleges of Great Britain and Ireland.

You will have seen that in order to be a successful investigator into tropical diseases a careful study of various subjects is required, amongst which I could mention parasitology, and particularly protozoology, as well as a thorough knowledge of tick and insect life generally. In the field of pathology a practical study of the blood lesions is essential, since nearly all tropical diseases are diseases of the blood, and for the purpose of the prevention of diseases and for the immediate help and saving of animals, the principles of vaccination and serum therapeutics are essential.

It is the duty, gentlemen, of you who are the governors of this institution to see that facilities are offered to give full attention to these problems, and of you who are the professors and teachers to enter thoroughly into them in your lectures. For you who are students it will be wise if you follow such lectures with the greatest care. The colonies require only the best of good men, and for such a great future is possible.

At the combined show of the Great Dane and Borzoi Clubs, held at the Crystal Palace on April 19 and 20, Mr. A. Cornish-Bowden, M.R.C.V.S., was selected for the position of judge of the Great Danes. His popularity amongst exhibitors of this variety, together with his reputation for fairness and knowledge of the points of the breed, gained for him no less than 391 entries, a record which has never been excelled, either in London or Berlin.

Miscellaneous.

UNIVERSITY OF LONDON.

Appointment of External Examiners in Veterinary Science.

Professor Sir John McFadyean, C.M., M.B., B.Sc., LL.D., has been appointed External Examiner in Veterinary Pathology, and Professor G. A. Buckmaster, M.D., B.Ch., D.P.H., External Examiner in Veterinary Physiology, for the year 1910-11.

ROYAL VETERINARY COLLEGE.

Election of Examiners, 1910.

Membership Examination—Class A.

Anatomy.—W. Robb, F.R.C.V.S.; John Blakeway, F.R.C.V.S.
Chemistry and Physics.—H. Somerville, B.Sc., M.R.C.S., L.R.C.P.; J. M. H. Munro, D.Sc., M.R.C.S., F.I.C., L.R.C.P.
Biology.—W. E. Agar, M.A., D.Sc., Fellow of King's College, Cambridge; W. B. Bottomley, Ph.D., M.A., F.L.S., F.C.S., Fellow of King's College, London.

Class B.

Anatomy.—H. G. Bowes, F.R.C.V.S.; W. E. Ison, F.R.C.V.S.
Histology and Physiology.—*E. P. Cathcart, M.D., D.Sc.
Stable Management, &c.—H. J. Dawes, F.R.C.V.S.; W. S. Mulvey, F.R.C.V.S.

Class C.

Morbid Anatomy, Pathology, and Bacteriology.—H. C. Reeks, F.R.C.V.S.; E. J. McWeeney, M.A., M.D., F.R.C.P.I.
Materia Medica, Practical Pharmacy, Therapeutics, and Toxicology.—James Peddie, F.R.C.V.S.; J. McL. Young, F.R.C.V.S., F.R.S.E., F.Z.S.
Hygiene and Dietetics.—W. Woods, F.R.C.V.S.; Henry Taylor, F.R.C.V.S.

Class D.

Veterinary Medicine and Meat Inspection.—W. H. Bloye, F.R.C.V.S.; N. Almond, F.R.C.V.S.
Veterinary Surgery and Obstetrics.—W. Hunting, F.R.C.V.S.; R. J. Hickes, F.R.C.V.S.

Fellowship Examination.

Veterinary Medicine and Surgery.—J. Macqueen, F.R.C.V.S.
Hygiene and Sanitary Science.—W. Hunting, F.R.C.V.S.
Pathology and Bacteriology.—J. Malcolm, F.R.C.V.S.

The examiners hold office for a period of three years.

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* Another to be appointed.

Translations.

A CASE OF DIAPHRAGMATIC HERNIA.

By Dr. STEINBERG.

Gelsenkirchen.

On August 12, 1909, I was called at 12 o'clock in the night to see a horse with the following history: At 7 o'clock in the evening the horse slipped when turning in the stall, and fell down. He immediately got up again, had one hour's work in a light waggon without showing anything abnormal, and was brought in again. At 8 o'clock he had eaten up his supper, and was put to work again. On the way back, about 10 o'clock, the driver found that the horse would not trot. He was walked home, and then showed, when stabled, all the symptoms of colic. The owner thereupon gave him a draught of "anticolicum." He dunged several times. When I arrived I found a light draught horse, aged about 10, with head depressed, and in a very apathetic state; there was strong in- and expiratory dyspnœa; at times groaning. Mucous membranes of the eyelids, looked at by lamplight, were pale. Pulse scarcely perceptible, irregular, and beating about 100 to the minute. Temperature not taken. The horse turned on his back, then lay on his side, sprang up, and then went down, and for some time assumed the dog-sitting posture. Whilst noticing him there were only slight colicky symptoms. Peristalsis was quite audible on both sides. On rectal examination the bowel was filled with loose dung, but nothing special was noticed.

An unfavourable prognosis, with probably an early death, was given; but, since the owner wished treatment, an arecolin injection was made and coffee administered. The pulse then became stronger. Death occurred at 2 a.m.

Post mortem on the morning of August 13: On opening the abdominal cavity nothing special was noticed in the intestines. The stomach was stretched and filled with masses of food. On the peritoneal surface of the liver there were a few small blood-clots. On removing the liver one noticed at the spot where the tendinous portion of the diaphragm merges into the muscular part, on the right side and in the upper third, an opening about as long as one's finger, in the partition through which a loop of the small intestine had gone into the thorax; this loop of intestine, about 1½ metres, was easily drawn back into the abdominal cavity. The edges of the rent were torn and covered with coagulated blood. The serosa of this piece, with the corresponding mesentery, was diffusely reddened and sharply defined from the remaining pale yellow-coloured bowel. The mucous membrane was swollen. The contents of this section were a thinish, dirty yellow-coloured fluid and much gas. Except for some filamentous efflorescences on the pleura pulmonalis there was nothing abnormal.

The rupture of the diaphragm doubtless resulted from the fall. The passing through of the intestine occurred after taking food, and perhaps the full stomach helped it. The chief symptoms *intra vitam* were: Strong dyspnœa, the depressed head, and the high frequency of the pulse, and slight colic appearances.

(*Berliner Tierärztliche Wochenschrift.*)

HYGIENIC PRODUCTION OF MILK.[1]

By MONSIEUR BRENET.

Veterinary Health Department, Seine.

Cowhouse.—The building, spacious, well lighted, well ventilated, in a perfect state of cleanliness, contains two rows of animals facing each other, but separated by a large service passage. Mangers of fortified cement have all their angles rounded, but are not separate. Walls cemented only up to a height of about a foot and two-thirds from the ground are carefully washed every day in their lower parts, and frequently limewashed for the rest of their extent.

Several movable tubs placed in the cleaning passages, which are also very wide, are destined to receive the excrements in proportion to their evacuation. The litter is plentiful, and renewed every day.

Isolation House.—A two-stall shippon, entirely independent of the cowhouse, is used to place fresh cows in; they remain there until the attendant veterinarian has declared them to be healthy. After arrival they are submitted to the tuberculin test; those that react are immediately returned to the would-be seller.

In this isolation building the cement mangers are separate; all the angles of the walls are rounded, the floor is strictly impermeable, without any breaks in it. The walls are cemented up to a height of 6 ft. 6 in. The cubic air space is considerable; the lighting and ventilation are exceedingly good, leaving nothing to be desired.

Choice of Animals.—The number of cows in the large house is twenty-one (twenty Normandy and one Flemish). They are all young and in perfect condition. Their price varies from between £25 and £33, and they are only kept six months in service. At the end of this time the dealer takes them back for a sum hardly equal to half the price at which they have been sold.

Ration.—Beetroot and all distillery and brewery residues are rigorously excluded from the feeding. The ration of a cow for one day is composed as follows: Fine maize meal, 4 lb. 6 oz.; barley meal, 2 lb. 3 oz.; lentil meal (at 10d. a kilogramme), 2 lb. 3 oz.; bran, 6 lb. 9 oz.; potatoes, 17 lb. 9 oz.; park hay, 22 lb. The cost of the food per day and cow comes out at about 4 francs, or 3s. 4d.

Milking.—Twice a day in a special well-fitted edifice, to which the animals are conducted in turn. The milkers, two in number, put on overalls specially cleaned each day. They wash the udder of the cow with tepid water and soap, then, after rinsing with fresh water, they spray the mammary region with oxygenated water with a vaporizer. They carefully wash their hands and put them in oxygenated water before proceeding to milk.

Manipulation of the Milk.—The milk is put into a large funnel of nickeled copper communicating directly with a cooler in the storey below. Filtering takes place through a cotton-wool disc fixed in a special way at the entrance of the apparatus.

Before each milking the cooler is carefully washed in boiling water, then sterilized with steam at 105° C. A very ingenious apparatus installed in the washhouse permits the obtaining of fresh boiling water at will, and steam under pressure.

[1] Visit to an establishment operating in the environments of Paris.

Washing Bottles.—Bottles destined to receive the milk are at first washed in carbonated baths at 80° C., are afterwards rinsed automatically in hot water, then sterilized by steam at 105° C.

Placed immediately in metallic boxes closing hermetically, they are put on a rack in the vicinity of the cooler. Immediately after filling the bottles are corked with a metallic capsule fastened to the edges of the neck by a mechanical catch.

This operation of corking costs about 4 centimes a bottle, but by a special arrangement on opening the bottle the capsule is ruptured into three parts, which cannot be put into position again, and render the covering of no further use.

This feature prevents all frauds which could be made by the deliverers of the milk to the prejudice of the establishment. The dairy sells the greater part of the milk in Paris, where it is sent by rail. The price of a litre (1¾ pints) has not been given to us; it should vary between 1 and 2 francs.

(*L'Hygiène de la Viande et du Lait.*)

PROLONGED ANÆSTHESIA BY A MIXTURE OF OXYGEN AND ETHYL CHLORIDE.

By PIERRE ROSENTHAL and ALB. BERTHELOT.

Ethyl chloride can only be used as a general anæsthetic in operations of short duration. When narcosis has to be prolonged it can only be effected by copious inhalations frequently repeated. Rosenthal and Berthelot thought it would be possible to prolong narcosis indefinitely by associating oxygen and ethyl chloride. The authors have been able under these conditions to maintain anæsthesia for nearly an hour, and to practise the most varied operations on animals. Narcosis is very rapidly obtained, and awakening is very prompt. Ethyl chloride has the advantage of not associating with operation shock the inconveniences of ether and chloroform.

(*Revue Générale de Médecine Vétérinaire.*)

INFECTIOUS BULBAR PARALYSIS IN DOGS.

By LAUFER.

Allatorvosi Lapok.

At a farm three dogs fell ill in the space of two days with the following symptoms: Strong salivation, rubbing of the head against neighbouring objects. Death occurred at the end of the first day of illness.

Autopsy disclosed no lesion in the organs. A rabbit inoculated under the skin with a piece of the spinal cord of one of the dogs was ill for two days and a half, and later showed typical trouble. A cat which ate the flesh of the rabbit was found dead nine days later.

The author has previously reported two great enzootics of the same disease in dogs which died at the rate of four or five a day.

(*Revue Générale de Médecine Vétérinaire.*)

Books and Periodicals, &c., Received.

Bulletins of The Bureau of Sleeping Sickness; Bureau of Animal Industry, U.S.A., 25th Annual Report; Report of the Municipal Veterinary Surgeon for Glasgow; The Commercial Motor; Journal of the Royal Army Medical Corps; Proceedings of the Royal Society of Medicine; Agricultural Journal of the Cape of Good Hope; Report of the Department of Agriculture for the Dominion of Canada.

Letters and Communications, &c.

Dr. Ebbitt; Professor Mettam; Mr. Kendal; Mr. P. R. Thompson; Mr. F. Bullock; Mr. A. N. Trotter; Entomological Research Committee; Mr. Douglas; Board of Agriculture and Fisheries; Department of Agriculture and Technical Instruction for Ireland; Messrs. Burroughs, Wellcome and Co.; Mr. P. J. Howard; Mr. Gelvin; Mr. J. G. Rutherford.

NOTE.—All communications should be addressed to 8, Henrietta Street, Covent Garden, London, W.C. Telephone, 4646 Gerrard. Telegrams, "Baillière, London."

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